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CONTENTS OF PREVIOUS NUMBERS

MAY, 1925. NUMBER 3

The Treatment of Infections and Infectious Diseases with Mercurochrome-220 Soluble. An Analysis of Two Hundred and Ten Cases That Furnish Many Definite Examples of a Therapia Sterilisans Magna. Hugh H. Young, M.D.,; J. H. Hill, M.S., and W. W. Scott, M.D., Baltimore.

Exhaustion. VIII. Anaphylaxis; Pregnancy. G. W. Crile, M.D., Cleveland.

Hypernephroma and Carcinoma of the Kidney; A Comparative Study. D. K. Rose, M. D., St. Louis.

Separation of the Upper Epiphysis of the Radius. Albert H. Montgomery, M.D., Chicago.

Embolectomy. Report of a Case. Arthur M. Dickson, M.D., Albany, N. Y.

Treatment of Epiphyseal Separation of the Lower End of the Radius. Isadore Zadek, M.D., New York.

The Effect of the Unit Record System and Improved Organization on Hospital Economy and Efficiency. M. W. Openchowski, M.D., New York.

The Deposition of Calcium Phosphate and Calcium Carbonate in Bone and in Areas of Calcification. James Crawford Watt, M.D., Toronto.

Abnormal Arteriovenous Communications. Acquired and Congenital. II. The Origin and Nature of Arteriovenous Aneurysms. Cirsoid Aneurysms and Simple Angiomas. Mont R. Reid, M.D., Cincinnati.

Twenty-Sixth Report of Progress in Orthopedic Surgery. Nathaniel Allison, M.D.; Philip D. Wilson, M.D.; Robert Soutter, M.D.; Harry C. Low, M.D.; Murray S. Danforth, M.D.; C. Herman Bucholz, M.D.; Lloyd T. Brown, M.D.; M. N. Smith-Petersen, M.D., and Robert B. Osgood, M.D.

JULY, 1925. NUMBER 1

Basal Metabolic Rates and Their Relation to End-Results in Thyroid Disease. A Statistical Study. Sara M. Jordan, M.D., Boston.

Appendectomy. A Study in Technic. C. A. Roeder, M.D., Omaha.

Abnormal Arteriovenous Communications, Acquired and Congenital. III. The Effects of Abnormal Arteriovenous Communications on the Heart, Blood Vessels and Other Structures. Mont R. Reid, M.D., Cincinnati.

Epithelioma of Cavities and Internal Organs of the Head and Neck. Albert C. Broders, M.D., Rochester, Minn.

Enchondroma of the Femur. Report of a Case with Repeated Recurrences and Ultimate Death. Robert G. LeConte, M.D.; Walter Estell Lee, M.D., and William P. Belk, M.D., Philadelphia.

The Relationship Between the Pathologic and Clinical Aspects of the Diseases of the Thyroid Gland. Henry C. Falk, M.D., New York.

Experimental End-to-End Anastomoses of the Colon. An Anatomic Study. Ferdinand C. Lee, M.D., Baltimore.

Injury, Including Irritation and Cancer. William M. L. Coplin, M.D., Philadelphia.

Reflex Spasm of the Pylorus and Its Relation to Diseases of the Digestive Organs. Walter Hughson, M.D., Baltimore.

A Review of Urology Surgery. Transplanted Muscle in Resection of the Kidney. Albert J. Scholl, M.D., Los Angeles; E. Starr Judd, M.D., Rochester, Minn.; Linwood D. Keyser, M.D., Roanoke, Va.; Gordon S. Foulds, M.D., Toronto, and Jean Verbrugge, M.D., Antwerp, Belgium.

AUGUST, 1925. NUMBER 2

Variations in Blood Sugar in Relation to Operation on the Thyroid Gland. Charles C. Lund, M.D., and E. P. Richardson, M.D., Boston.

The Injection of Absolute Alcohol into the Thyroid Gland. Experimental Observation of a Suggested Clinical Procedure. Howard B. Barker, M.D., Ann Arbor, Mich.

Dyspnea Following Thyroid Operations. Ralph Colp, M.D., and Henry W. Louria, M.D., New York.

Osteoplastic Skeletal Metastases from Carcinoma of the Breast. Report of an Unusual Case. Solomon Ginsburg, M.D., New York.

Abnormal Arteriovenous Communications, Acquired and Congenital. IV. The Treatment of Abnormal Arteriovenous Communications. Mont R. Reid, M.D., Cincinnati.

The Secretion Antecedents and Mitochondria in Pathologic Thyroids. A Theory of the Mode of Secretion in the Thyroid Gland. J. Albert Key, M.D., St. Louis.

A Review of Urologic Surgery. Albert J. Scholl, M.D., Los Angeles; E. Starr Judd, M.D., Rochester, Minn.; Linwood D. Keyser, M.D., Roanoke, Va.; Gordon S. Foulds, M.D., Toronto, and Jean Verbrugge, M.D., Antwerp, Belgium.

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ARCHIVES OF SURGERY

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CHRONIC SUBDURAL HEMATOMA

ITS PATHOLOGY, ITS RELATION TO PACHYMENINGITIS HEMORRHAGICA AND ITS SURGICAL TREATMENT *

TRACY JACKSON PUTNAM, M.D.

WITH CASES CONTRIBUTED BY MEMBERS OF THE SOCIETY OF NEUROLOGICAL SURGEONS AND A PREFATORY NOTE BY

HARVEY CUSHING, M.D.

BOSTON

PREFATORY NOTE

Since the foundation of the Society of Clinical Surgery some twenty years ago, there has been a tendency for other limited groups of clinicians who have common problems, sympathies and interests to band themselves into migratory organizations which meet in rotation at one another's place of work. One of the youngest of these is the Society of Neurological Surgeons.

Certain members of this society in their desire to advance information concerning a poorly understood and supposedly rare intracranial lesion have generously pooled their experiences in regard to it, so that a single individual might have at command sufficient material for a comprehensive study. Whether such a novel and unselfish program will lead to anything beyond the present report cannot be foreseen, but it at least sets a good precedent. Certainly such a composite of experiences is of greater value and will arouse wider interest than the mere presentation of the subject from the standpoint of a single clinic. It must be borne in mind, however, that those who have been contributors of material for this purpose cannot be held responsible for Dr. Putnam's comments and conclusions, to some of which they may quite possibly take exception.

Aside from the unhappily vivid mental picture of one unfortunate experience with a subdural hematoma under circumstances to be described, I confess to having had only the most vague recollections of previously encountering at operation any considerable number of the lesions under consideration. It consequently has been a surprise and the source of some mortification to find how many examples are buried under various titles in our past case histories. Though mindful of Wilfred Trotter's paper on the subject with the report of four cases published in

* From the Surgical Clinic of the Peter Bent Brigham Hospital.

1914, the war had dimmed even the memory of this excellent article, and my ideas regarding the relation of the old time pachymeningitis hemorrhagica interna of Virchow to these posttraumatic and essentially surgical lesions were highly confused.

When the case with which Dr. Putnam introduces his report was presented to the society, particular attention was drawn to the peculiar greenish appearance of the dural surface of the partly organized clot. This peculiarity seemed to me to be the one feature of the case that was unusual and unaccountable.

As chance would have it, two other patients with the condition were admitted to the Peter Bent Brigham Hospital shortly after the time of the meeting. Our attention having been newly drawn to the subject, the nature of the process was correctly surmised before operation in both these cases. When the dura was exposed, the two cases, furthermore, showed the same unmistakable greenish color of the subdural clot through the semitranslucent dura. Though a discoloration of the dura of some sort is commonly observed in these cases, it is variously described. Drs. Sachs and Naffziger, for example, speak of the membrane in their cases as having shown a bluish tint. Since Dr. Putnam lays no special emphasis on this matter and since the clot in the first three cases he reports was so green as strongly to suggest the presence of bile pigment, the subject may deserve brief consideration in this prefatory note.

Due to a series of important studies on hepatic function, beginning with those first undertaken twelve years ago by G. H. Whipple and C. W. Hooper in the Hunterian Laboratory of the Johns Hopkins University Medical Department and culminating in the recent experiments of F. C. Mann and his co-workers at the Mayo Clinic, the idea of an extrahepatic formation of bile has come to prevail. It is believed, in short, that the transformation of hemoglobin into biliary pigments is a function of the endothelial clusters widely distributed in the body, of which the Kuppfer type is the best known, and that the hepatic cells merely serve as an excretory mechanism for bile which is preformed elsewhere. This apparatus, which appears to have been first described by Goldmann, is the reticulo-endothelial system of Aschoff.

The cells of this system doubtless have a multiplicity of functions. One of these functions is the metabolizing of hemoglobin in the blood stream, but the cells being migratory and phagocytic also play an active part in the breaking down and disposal of extravasated blood elsewhere. So it is that in old blood clots, wherever situated, the presence of bile salts may usually be demonstrated. But so far as I am aware, it is only on the surface of the subdural hematomas that a sufficient amount of bile pigment is deposited to produce the deep icteric color under consideration.

It is of interest to speculate on the cause of this tendency of a subdural hematoma to become bile stained. Though Aschoff does not include the mesothelial cells of the arachnoid in his reticulo-endothelial system, we know from the studies of L. H. Weed and C. R. Essick that they represent an apparatus capable of marked local reactive phagocytosis. And in our present connection it is of interest that these authors found the subarachnoid injection of partially hemolized red corpuscles to be the most favorable means of so activating these cells that they assume the characteristics of ameboid macrophages. Moreover, S. C. Harvey has shown that they play the essential rôle in reparative processes. It is due, for example, to the rapid multiplication of these cells that meningeal defects are quickly healed and that foreign substances embedded in the meninges become encapsulated by a smooth, glistening membrane. But there is no better example of their capacity in this regard than the prompt and efficient way in which a subdural hematoma becomes encapsulated.

It consequently is safe to assume that these mesothelial cells of the leptomeninges are in their action similar to if not identical with the cells of the reticulo-endothelial apparatus as it has been described by pathologists in other parts of the body, more particularly in the liver and spleen; and as a part of this apparatus that they possess among other properties the ability to transform hemoglobin into bilirubin and biliverdin. These subproducts, indeed, become so heavily deposited on the surface of a long standing subdural hematoma as to give it an unmistakable icteric color. On the other hand, when the clot has been removed the subpial surface of the compressed brain will usually be found to have the yellowish color commonly observed in old cerebral extravasations of any sort, owing, as Virchow long ago showed, to the presence of hematoidin crystals.

Unfortunately, no tests were made of the outer layer of any of these greenish surfaced clots with the end in view of demonstrating the presence of bile salts. My colleague, Dr. Berglund, however, has from time to time examined the dark, greenish fluids obtained from some of the intracranial tumor cysts, and has demonstrated that the color is due to biliverdin. All this, to be sure, deals with the extrahepatic formation of the biliary pigments which is a matter remote from practical surgery, and, after all, it is in the practical applications of this present study to the operative treatment of these hematomas that the surgeon will be chiefly interested.

In his review of our hospital histories, Dr. Putnam has come on the records of the six examples that he has included. But having personal memory of one or two other cases that I thought might shed some further light on the subject, I have found records of six additional cases, variously diagnosed and indexed under such general headings as intra-

cranial hemorrhages. Three of these cases were ones in which extensive, subpachymeningeal pseudomembranes were disclosed during exploratory operations for suspected frontal lobe tumors: one many years after trauma; another in an alcoholic patient; and another a year after a cranial trauma in a child, whose blood incidentally showed a double plus Wassermann reaction. Whatever may have been the etiologic factor in these three cases, the other three were frankly of the sort under consideration, and one of them perhaps deserves being added to the present report because of the unusually short latent interval between the reception of the insignificant trauma and the onset of pressure symptoms produced by the clot—a history very similar to that of the more common extradural hematoma from a torn meningeal artery. The story is one that brings to mind a most painful clinical experience.

Miss I., aged 68, the principal of a school near Philadelphia, a woman in perfect health, during the Christmas recess of 1914 had been on a visit to some friends in Boston. December 28, about midday and shortly before her departure, she slipped on a rug and fell backward, bumping her head in the right parietal region. Though momentarily stunned, she made light of the matter, finished her packing and in a couple of hours boarded her train. A severe headache soon set in and by the time Providence was reached at 3:30 p. m., she had become semiconscious and was removed from the train to a local hospital. There she is said to have shown some aphasia and to have had some twitching of the right arm. The following day, though confused, she appeared to be somewhat better; but December 31 she became stuporous, acquired a rhythmic type of respiration, and in this state was brought back to Boston.

On her admission to the Brigham Hospital, she was in a deep stupor with pronounced Cheyne-Stokes respiration. Great restlessness coincided with the periods of hyperpnea. At these times, all the extremities were moved, the right apparently less well than the left. There also was a definite obscuration of the left disk. The cerebrospinal lumbar fluid was in all respects negative.

It was assumed that she probably had an extradural hemorrhage which, in view of the story, in all likelihood lay on the left side. Without anesthesia a subtemporal exploration was made. It revealed a slightly tense brain with no ecchymoses or other evidence of contusion in the normal appearing meninges.

It was thought that an erroneous diagnosis had been made and that the symptoms must have been produced by some deep seated lesion. The patient's condition remained practically unaltered, though on the third postoperative day there was a slight return of consciousness for a few hours. The Cheyne-Stokes respiration, with stupor deepening to coma, persisted to the end, which occurred January 7, ten days after her fall.

The necropsy showed a large subdural collection of semifluid, dark blood clot, greatly deforming the unexplored (right) hemisphere. The clot was slightly adherent to the dura where organization had apparently begun. The pia arachnoid over the flattened brain was discolored but intact. The source of the bleeding was not determined.

Grievous though it is to recall this story, it is one that gives a lesson as to the desirability of bilateral exploratory punctures under similar circumstances, a procedure that Dr. Putnam wisely advocates.

There can be no doubt but that the files of all general hospitals must contain many records of subdural hematomas which perhaps, as in this case, have been variously indexed, and so are difficult to trace. However, in view of the fact that the majority of these hematomas, unlike the case given above, are so slow in their formation as to bear no apparent relation to any recalled injury, it is not at all surprising that the psychoses which commonly ensue make the finding of organized subdural hematomas far more common at necropsy in psychopathic than at operation in general hospitals.

Dr. Putnam's report serves to show several things: that these subdural hematomas are far less rare than is usually supposed; that in the majority of cases, and perhaps in all, they follow on a trauma so insignificant as to be commonly forgotten by the patient or overlooked in the anamnesis; that the diagnosis should always be considered in a history of mild trauma followed after a latent interval, often of surprising length, by severe headaches associated with psychoses; that, a correct diagnosis being made, the indications for operation are as definite as those for the well recognized extradural hemorrhages associated with fracture of the skull; that perfect recovery is to be expected on evacuation of the clot; that continuance or recurrence of the bleeding is unusual, and, finally, that one's ideas of so-called pachymeningitis hemorrhagica interna with its supposed spontaneous and successive bleedings may possibly have to be entirely recast.

HARVEY CUSHING.

INTRODUCTION

At a meeting of the Society of Neurological Surgeons at the Peter Bent Brigham Hospital, Dec. 7, 1923, the following case was shown:

CASE 1.—J. K., a school boy, aged 18, was referred to the hospital, November 9, by Dr. E. Wyllys Taylor of Boston.

At the age of 7 he had had a bad fall, striking the left side of his head, but with no apparent ill consequences. He grew up to be a bright, normal child. At the age of 12 he was found unconscious in a convulsion at the foot of a flight of stairs. Three years later, he had six attacks of a *grand mal* type within a year, with many lesser attacks between, sometimes amounting only to a momentary loss of consciousness. Since then, attacks had come less often, but at irregular intervals. His physician, Dr. Taylor, could find nothing objectively wrong to account for the seizures, and felt that the patient's personality was not that of an epileptic. The boy had continued to stand at the head of his class in school, and to be extremely active in athletics.

October 20, nineteen days before his admission, he was struck on the head and knocked unconscious for a few minutes while playing football. He was able to continue with the game, however, and felt well the next day. He played again three days later, and during the course of the game "bunted" another player with his head. That evening he complained of being tired, and of a severe headache in the left frontal region. During the night and morning, he vomited several times. The headaches continued throughout the subsequent course of his illness, and he vomited almost daily. November 8, he began to

have difficulty in naming objects and Dr. Taylor, finding that he had a choked disk, referred him here.

On admission the following day, he was seen to be a well developed but rather thin and pale boy, lying in bed complaining of headache. He had a bilateral choked disk of 3 diopters, perhaps a little more on the left. There was slight weakness of the left abducens. The right hand displayed some awkwardness, and at times the deep reflexes were exaggerated on the right side. The right abdominal reflex was absent.

There was a definite aphasia. He could recognize objects, and describe or illustrate their use, but found difficulty in naming them. He showed marked perseveration, repeating "I can't seem to get it—I can't do a thing." Examination was otherwise essentially negative.

The pulse was slow, from 50 to 70, and he had a slightly irregular temperature with an evening elevation to 99 degrees and a subnormal (once 95.2 degrees) morning registration. Routine laboratory tests, including visual fields, stereoscopic roentgen rays, etc., were essentially negative. A lumbar puncture was performed, November 14, and 10 c.c. of slightly yellow fluid, under approximately normal pressure, was withdrawn. It showed no pleocytosis. The Wassermann reaction in it and in the blood serum was negative.

After his admission he at first showed improvement but, November 15, he had a severe generalized convulsion, starting on the right side. This episode and the fact that the choked disk failed to subside led to an exploration of the left hemisphere. The diagnosis lay between tumor and intracranial hemorrhage.

November 19, under ether, Dr. Cushing turned down a large bone flap in the left frontoparietal region. The diploe was unusually soft and thick. When the flap was reflected, the dura was found to be under considerable tension and of a greenish hue. When it was incised, a peculiar olive-colored, gelatinous structure was exposed, from which the dura peeled smoothly and with little bleeding. The greenish surface extended in all directions over the hemisphere, disappearing under the margins of the reflected dura. The lesion proved, on incision, to be an old, partially liquefied clot about 2 cm. in thickness, on the lower cerebral surface of which was another friable membrane about the thickness and consistency of wet blotting paper. This lay on the arachnoid without being in the least attached to it. When it was brushed away, a compressed and yellowish but otherwise normal cortex was exposed. The relations of the various membranes and their characteristic colors are illustrated in the sketch begun at the time of operation (Fig. 1).

The central and exposed area of clot, measuring about 6 by 7 cm., was then lifted away, and it was possible to pull out large pieces of thinner marginal clot from beneath the edges of the dura. The more inaccessible and thin fragments, which extended well down under the temporal lobe, were not detached. As there was considerable oozing, much time was consumed in a painstaking toilet of the wound until hemostasis appeared to be complete. The dura was then resutured and the bone flap returned to its position without a drain.

The patient made a good operative recovery; his aphasia was less marked the morning after the operation than before, but three days later he had a sudden exacerbation of headache with greatly increased difficulty in speech. It was feared that the clot might have reformed and for a time the advisability of a reexploration was considered. Other than for this temporary upset, he had a smooth convalescence until December 8, the day before his discharge, when he was to be shown before the Society of Neurological Surgeons. On the way to the clinic, he had a brief generalized convulsion. The following morning he felt perfectly well and was allowed to go home. Both disks were flat. There

Fig. 1 (Case 1).—A bone flap has been elevated and a dural flap reflected. Beneath is seen the greenish brown clot, which is incised to show its thickness, revealing the cortex covered by normal arachnoid. (From a sketch made at time of operation.)

was no speech or memory defect and no residual motor or sensory disturbance.

Dr. Taylor reports that the patient has continued having mild convulsive attacks similar to those he has had for so many years, but aside from this has remained for the last year in perfect health.

Microscopic sections taken through both membranes detached from the dura showed an old clot composed of red cells in various stages of disintegration caught in the meshes of a heavy fibrin network. Only occasional leukocytes were seen. On the surface toward the arachnoid, there was a definite mesothelium made up of large, spindle shaped cells with pale, oval nuclei, lying on a continuous membrane of fibrous tissue, six or seven cells in thickness (Fig. 2). It contained no blood vessels. It lay directly under the most fresh looking portion of the clot, which in preparations stained for connective tissue was seen to be still unorganized.

Along the surface that was stripped from the dura, organization was most advanced. Here another much thicker membrane was seen, composed of dense, cellular connective tissue, through which ran many rather coarse capillaries (Fig. 3).

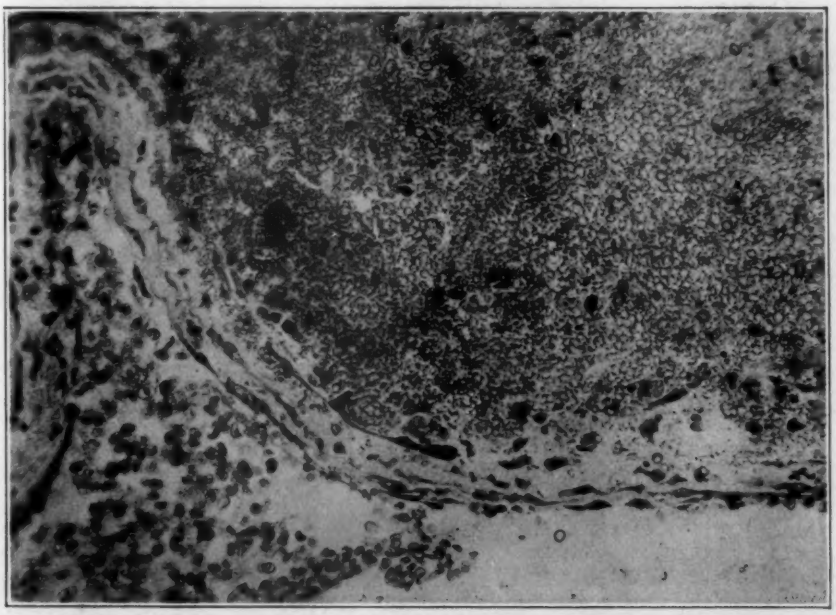

Fig. 2 (Case 1).—Thin neomembrane underlying clot; membrane lined with mesothelium on the side toward the arachnoid; stained with methylene blue and eosin; x 300.

A striking feature of this region was the presence of several rounded and irregular spaces, measuring up to 40 microns in diameter. They were lined with flat mesothelium, which was sometimes discontinuous and contained few or many red cells, a small amount of fibrin, and granular debris. No fibrin was seen in the capillaries. There was considerable interstitial hemorrhage, perhaps from the trauma of removal. A sharp but irregular line of demarcation lay between the firm granulation tissue and the well preserved clot, which contained only a few strands of collagen and no vessels. The thickness of the organized

layer was extremely variable, however, and in places large bays or lakes of clot, usually containing little fibrin, were entirely surrounded by connective tissue.

No bacteria were seen in sections stained with methylene blue and eosin.

Summarizing, a boy, aged 18, subject to occasional epileptiform attacks but otherwise normal and healthy, developed headache, aphasia, and choked disk seventeen days after a mild trauma to his head. At operation, nine days later, an

Fig. 3 (Case 1).—Membrane of granulation tissue stripped from dura. The line of dural attachment is seen at the top. A little way below are a few large, rounded spaces, mostly empty. Below them is a zone of well formed connective tissue containing capillaries with slight extravasation about them. The uneven, irregular character of the clot is seen at the bottom of the picture; phosphotungstic acid hematoxylin stain; x 80.

organizing clot was found beneath the dura mater over almost the entire left hemisphere. It was removed, and the patient in course of time made a complete recovery.

At the time the case was presented, Dr. Cushing inquired of the visitors whether they had encountered similar conditions. Several had seen them, and Drs. Adson, Naffziger and Sachs very kindly supplied full notes of one or more cases each. Dr. Heuer had already published one.[1] Since then three other cases under the care of Drs. Horrax and Cushing have been observed in this clinic. In addition, the records of two earlier cases, one of which resulted in a fatality, have been found. This paper may be said, therefore, to be prepared for the Society of Neurological Surgeons.

Scope of the Present Study.—It is not easy to define the cases to be considered in the present work except in clinical terms. There appears to be a definite group of patients who develop symptoms of intracranial pressure, local or generalized, from several days to several months after a blow on the head, or rarely without history of trauma, who are found to have an organizing blood clot on the inner surface of the dura. Many of these cases have been reported in surgical journals as late or secondary subdural hemorrhages; others have been recorded, mainly in the pathologic literature, as a subvariety of pachymeningitis hemorrhagica interna. It is difficult to draw a hard and fast line between the two, and a discussion of both will be required to throw light on the nature of the disease outlined above. We may arbitrarily limit our chief interest to those cases in which the signs of pressure develop a week or more—allowing time for organization to begin—after trauma, and we may give only passing attention to those cases in which the process is of such a low grade, or so masked by other diseases, that operative intervention is not to be considered. In addition, the indications for, and results of, surgical treatment will be reviewed.

HISTORICAL REFERENCES

As with many debated subjects in medicine, it is difficult to form a true estimate of the various hypotheses which have been advanced to account for subdural hemorrhages without understanding the circumstances under which they were framed. Different schools and nationalities each have their own interpretation of the phenomena observed, and pathologist, experimenter and surgeon each has his own class of material for study.

One of the first recorded cases of subdural hemorrhage in history is that of Henry II of France, who died in 1559, following a supra-orbital wound received in a tourney, according to Paré.[2] This was an acute hemorrhage, however, and no operation was attempted.

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1. Heuer, G., and Dandy, W.: A Study of 100 Consecutive Cases of Brain Tumor, Bull. Johns Hopkins Hosp. **27**:224-237 (Aug.) 1916.

2. Probst, M.: Ueber Pachymeningitis cervicalis und über Pachymeningitis interna haemorrhagica bei chronisch fortschreitenden Verblüdungsprocessen in der Jugend, Arch. f. Psychiat. **36**:114-143, 1902.

Subdural hemorrhages without precedent trauma were, naturally, observed only after the opening of the era of pathology. In 1657 Johannes Wepfer performed a necropsy on a patient of 70 who died some hours after an "apoplectic" stroke, with aphasia and hemiplegia.[3] He was assisted at the postmortem by Harderus. A bloody cyst about the size of a hen's egg was found beneath the dura. He believed that he could demonstrate multiple ruptures of the meningeal artery. Morgagni,[4] in 1747, reported a similar case in which the apoplectic symptoms were of several days' standing. He expressed some doubt as to the nature of the sac in which the old clot was found, but concluded it was composed of arachnoid. In neither of these cases was there any history of trauma.

During the first half of the last century, numerous cases of the same sort began to be reported, mostly by French and English pathologists. Good reviews of the early literature[5] are given by Schuberg[6] and by Charcot and Vulpian.[7] The first important paper is that of Houssard,[8] who in 1817 described the clot and the membranes surrounding it, which he believed were composed of fibrin. Bayle[9] in

3. Wepfer, J. J.: Observations Anatomicae ex Cadaveribus eorum, quos sustulit Apoplexia, Amsterdam, 1681, p. 5.

4. Morgagni, J. B.: De Sedibus et Causis Morborum, Ed. Lovanii, 1767, Lib. 1, Epist. 3, Art. 20.

5. These earlier reviews, valuable as they are, do not apply the rigid criticism that seems necessary nowadays to the cases reported in them. Thus, the work of Serres (Nouvelle division des apoplexies, Annuaire méd.-chir. d. hôp., Paris, **1**:246, 1819) to which references are so often given was concerned solely with leptomeningitis, as far as one can now understand his descriptions. Certainly none of his cases resembled those at present under discussion. On the other hand, his experiments were of considerable interest, though seldom noticed. The famous case of Bouillon-Lagrange (Observation d'hémiplegie du coté droit, produit par un épanchement sanguin enkysté à la surface de l'hémisphere droit du cerveau; lesion constatée six mois après la guérison, dans une autopsie judiciare fait à l'occasion d'un homicide, Arch. gén. de méd., Paris, **14**:313, 1847) in which the lesion was found at necropsy in a man who was murdered six months after recovery from a hemiplegia, should probably be excluded also, as the author definitely stated that the large hemorrhagic cyst that he observed was adherent to the visceral, and free from the parietal, arachnoid. Few of the early histories can be accepted without reserve.

6. Schuberg, W.: Das Haematoma Durae Matris bei Erwachsenen, Arch. f. path. Anat. **16**:464-512, 1859.

7. Charcot and Vulpian: Sur les néomembranes de la dure-mère, Gaz. hebd. de méd., Paris, **7**:728; 789; 821, 1860.

8. Houssard, M.: Observation d'un kyste considerable, developé dans la cavité de l'arachnoide chez un subject qui a succombé avec les symptomes d'une apoplexie sanguine, Biblioth. méd., Paris, **55**:67, 1817, quoted by Schuberg (Footnote 6).

9. Bayle, A. L. J.: Traité des maladies du cerveau et de ses membranes. Paris, 1826, p. 250.

1826 described twelve cases and first pointed out the similarity between the skin, organized membranes on the inner surface of the dura and the membranous envelops containing fluid or clotted blood. He believed that the lamination occurring in some of his cases was due to repeated hemorrhages. He placed the seat of the hemorrhage between the dura and the parietal layer of the arachnoid postulated by Bichat.[10] In the same year, Calmiel [11] also published observations on the subject. He was impressed with the coexistence of other manifestations of intracranial inflammation encountered in such cases, and apparently believed the subdural membranes were the product of a "periencephalitis." A series of papers by other pathologists, mostly in France, appeared during the next quarter century but, owing to lack of microscopic examination and to the confusion between the dura and the arachnoid, few of them are of value as case reports, and most of the theories advanced are stated in terms which are now unintelligible. Cruveilhier [12] and Heschl [13] definitely believed that an inflammatory membrane preceded the hemorrhage, while Baillarger [14] and Prescott Hewitt [15] reported cases in which there was good reason for believing that the membranous covering was the result of the effusion of blood, and the surrounding structures appeared quite free from inflammation. The latter believed that the extravasation might be caused by trauma, by a determination of blood to the head, or by vascular or cardiac disease.

Virchow's [16] classical paper was published in 1857. It is an important landmark, not only on account of the eminence of its author, but also because it is the first article to give a clear account of the histology of

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10. This is an error that has been committed by subsequent authors until very recently (Grandmaison, F.: Les méningitis cérébrales hémorrhagiques, Gaz. d. hôp., Paris, **70**:289-295, 1897; Francais; Gros hematome enkysté de la dure mère, Rev. neurol., Paris, **37**:759-761, 1921). Probably the appearance of a subdural false membrane confirmed the idea of a parietal arachnoid in Bichat's mind, for he stated: "I have seen the arachnoids in a subject that had been affected with a chronic inflammation evidently thickened on the internal surface of the dura mater, without this having experienced the slightest alteration; it was detached without difficulty, and torn with great ease" (Bichat, X.: General Anatomy, Hayward's translation, Ed. 8, Boston, 1822, **3**:141).

11. Calmiel: De la paralysie consideré chez les alienés, Paris, 1826, quoted by Schuberg (Footnote 6) and by Charcot and Vulpian (Footnote 7).

12. Cruveilhier, J.: Traité d'anatomie pathologique, Paris, **3**:515, 1856.

13. Heschl, R.: Kompendium der allgemeinen and speciellen Pathologischen Anatomie, Vienna, 1855, p. 291.

14. Baillarger: Du siège de quelque hémorrhagies meningées, Thèse de Paris, 1837.

15. Hewitt, Prescott: On Extravasations of Blood into the Cavity of the Arachnoid, Tr. Med.-Chir. Soc., London, 1845.

16. Virchow, R.: Haematoma Durae Matris, Verhandl. d. phys-med. Gesellsch. zu Würzburg **7**:134-142, 1857.

the membrane, and to suggest a plausible explanation in modern terms. So profound has been its effect on pathologic opinion up to the present day that it will be worth while to devote some space to a detailed consideration of it, and of Virchow's statements on the same subject in a later work.[17]

Virchow began with a well founded criticism of the supposed site of the hemorrhage, and pointed out that it does not lie within the dura nor under the "parietal" arachnoid, the existence of which he himself had helped to disprove. He recognized the possibility that a subdural hemorrhage might occur from trauma to large vessels, or might burst through the pia from the cerebral cortex, or might even occur spontaneously in hemorrhagic, valvular or vascular disease. But the structure formed in such a way, he believed, probably would not reproduce the appearance or behavior of the spontaneous hematoma. He did not describe any histologic difference between the two, but supposed that there must be one because fresh points of hemorrhage are seen in even old membranes, which suggests that they are progressive, and the clinical symptoms are chronic and intermittent.

The sequence of events he believed to be as follows: In certain patients, especially in the insane, the dura becomes chronically inflamed, as is shown by the exudation of a thin film of fibrin, sometimes tinged with blood, over its inner surface. Capillaries grow into this from the sparse vessels of the dura and, if conditions of its formation repeat themselves, another layer of fibrin may form and become organized. The new tissue may be so vascular "that scarcely any other form of tumor is to be compared with it." The vessels are very large, thin walled and irregular. Under the influence of hyperemia, they may rupture, producing small ecchymoses which absorb, leaving pigment behind them; or the separation of layers of the membrane may tear other capillaries and give rise to massive hemorrhages. These in turn become similarly organized and their capillaries may rupture. He gave the name pachymeningitis interna chronica to the fibrinous exudation and its organization; if extravasation were a prominent feature of the tissue, he called it pachymeningitis hemorrhagica. The bloody sacs or cysts he called hematomas of the dura. He recognized that the hemorrhagic contents of the cyst might become replaced by serum (hygroma of the dura), and considered this one of the forms of external hydrocephalus. He believed that small extravasations might regress but that large ones were almost invariably fatal.

As we shall see, Virchow's description of the process was accurate, and has merely been amplified by later histologists. But the appearances he described are certainly open to other interpretations, as has

17. Virchow, R.: Die Krankhaften Geschwülste, Berlin **1**:140, 1863.

often been pointed out. In the first place, the exudation of fibrin, and its simple organization does not necessarily constitute an inflammation. As Barrett [18] remarks, it certainly is not inflammation in the sense that pneumonia is an inflammation. But this is scarcely more than a difference of terms. In the second place, the appearance of well preserved blood corpuscles in an old membrane need not mean that they are the result of a fresh hemorrhage, for red cells may remain well preserved for weeks or even months in hematomas in other situations. The variability of symptoms also is an unreliable guide to the appearance of the membrane. In the third place, he does not describe the events which follow a traumatic subdural hemorrhage, and possibly never had encountered one, so the distinction he draws between the subdural hemorrhage and hemorrhagic inflammation of the pachymeninx must be regarded as theoretical. If a hemorrhage into an inflammatory membrane can organize and the new-formed vessels can rupture from hyperemia, one would expect that the same sequence of events would be possible as the result of a traumatic hemorrhage.

Virchow's views were accepted at once in Germany, and with some modifications are still orthodox there, in spite of the opposition of a few pathologists, such as Sperling,[19] Huegenin,[20] Buss [21] and Kasemeyer.[22] They were introduced into France by Charcot and Vulpian,[7] and have remained almost unchallenged in that country. British pathologists have mostly clung to the "hemorrhagic" theory.

An important paper was published by Kremiansky [23] in 1868, on the basis of a long series of cases encountered in a general hospital at St. Petersburg. He believed that the middle meningeal artery was always engorged in these cases, and that any increase in blood pressure would be most strongly felt by the capillaries nearest the main trunk, namely, in the parietal region, which was the location of the lesion in all the cases reported up to that time. He therefore thought that engorgement of the meningeal artery, produced by a general disease, was the cause of the fibrinous exudate and vascular organization tissue; but he also

18. Barrett, J. O. W.: On Pachymeningitis Haemorrhagica Interna, Brain **25**:181 (Part 2) 1902.

19. Sperling, H.: Ueber Pachymeningitis Haemorrhagica Interna, Inaugural Dissertation, Königsberg, 1872.

20. Huegenin, O.: Inflammation of the Dura Mater, Ziemssen's Cyclopedia, American translation, **12**:386, 1877.

21. Buss, O.: Zwei Fälle von Pachymeningitis interna haemorrhagica nach Trauma, Ztschr. f. klin. Med. **38**:451, 1899.

22. Kasemeyer, E.: Ueber post-traumatische Pachymeningitis unter dem Bild der post traumatischen Neurosen, Friedreich's Bl. f. gerichtl. Med. **62**:293-319; 339-384; 401-416, 1911.

23. Kremiansky, J.: Ueber die Pachymeningitis interna haemorrhagica bei Menschen und Hunden, Arch. f. path. Anat. **42**:129-161; 321-351, 1868.

recognized the possibility of local irritation (as from caries of the skull or a contiguous apoplectic focus) producing similar, milder changes. In two cases, he saw the organization of a traumatic subdural hemorrhage, resulting in a small, firm, unlaminated scar; but he gives no histologic description of the final lesion. To explain the occurrence of hemorrhagic dural cysts after a hemorrhage, he supposed that the presence of clot on the dura might in itself be irritating.

In a later part of the same paper, Kremiansky analyzed the clinical histories from the etiologic point of view, and found that the lesion occurred in patients suffering from many different kinds of infectious diseases, especially rheumatism and typhoid fever, but that in all cases there was a history of chronic alcoholism, which he believed was the fundamental cause. He conducted experiments on dogs, feeding them brandy daily for two or three months, and found a pachymeningitis in three out of four experiments.

Founded on an impressive series of cases, and with elaborate descriptions of numerous varieties of gross appearances, Kremiansky's article has exercised much influence on subsequent opinion. His microscopic descriptions, however, are not nearly as clear as Virchow's. Subsequent observers have, for the most part, failed to confirm the engorgement of the middle meningeal artery. Cases have been described in which typical membranes were found in the basal, frontal and even cerebellar regions.[24] No conclusive reason has been given why a subdural clot should be more irritating than a subcutaneous clot. The high incidence of subdural membranes among alcoholic patients is indeed striking, but many cases have now been reported in which alcoholism, and indeed any systemic disease, could be excluded. His experiments on dogs have been repeated by a number of observers, and subdural membranes have sometimes been produced, but in a far smaller proportion of cases.[25]

The inaugural dissertation of Sperling[19] appeared in 1872. In a concise review of the literature, he sought to show that an origin of the neomembranes from capillary or venous hemorrhages had never been excluded in the published cases. He then reported a series of experiments in which whole blood was injected beneath the dura, with the resulting formation of a membrane resembling the spontaneous lesion in many respects. He believed that this settled the question of the origin of all subdural membranes. A criticism of his experiments with those of van Vleuten,[26] which were interpreted in an exactly opposite sense,

24. Ciarla, E.: Beitrage zur pathologische Anatomie und zur klinische Studium der Pachymeningitis cerebralis haemorrhagica, Arch. f. Psychiat. **52**:439, 1913.

25. Saltykow: Referat, Alcoholismus chronicus, Centralbl. f. allg. Path. u. path. Anat. **22**:849, 1911.

26. Van Vleuten, C. F.: UeberPachymeningitis haemorrhagica interna traumatica, Inaugural Dissertation, Bonn, 1898.

will be reserved for another paper. It seems justifiable to say here that the problem of pachymeningitis cannot be solved by experimental means alone.

In writing the section on inflammation of the dura mater in Ziemssen's Cyclopedia, Huegenin [20] reverted to the "hemorrhagic" theory of origin. His description of the process corresponds closely with Virchow's; but for Huegenin, the thin fibrinosanguineous subdural layer was a hemorrhage and the resulting vascularization was merely the organization of a clot. He agreed that subsequent hemorrhage could occur from rupture of dilated capillaries, and that it was invariably within the membrane previously formed. Reviewing the changes occurring in the dura and elsewhere in the body, he concluded that they were inconstant and not in accord with the assumption that the pachymeninx was inflamed.

Huegenin's discussion of etiology is particularly valuable. He frequently found fatty degeneration and sometimes thrombosis of the cerebral veins in the pia and at their entrance into the sinus. Once he found a rupture of one of them in the pia, and once near the sinus. He found similar changes in the veins of the pacchionian granulations. He recognized the possibility of a traumatic origin, and referred to several cases, some original, in which the disease seemed due to trauma. He encountered a significant proportion of cases in patients suffering from hemorrhagic diseases, such as pernicious anemia, and in patients with heart disease.

Near the close of the last century, a series of papers appeared which again approached the question from a histopathologic point of view. The first of these was by Melnikow-Raswedenkow.[27] On the basis of preparations stained for elastic tissue, he believed he could demonstrate that all the pathologic changes in the dura took place in a "pachymeningitic zone" between the endothelium and a constant membrana elastica interna. As a result of toxemia, this zone became widened and vascular. He thought that the elastic membrane played an important part in the production of pachymeningitis, in that it acted as a partial barrier to the return of blood from the capillaries. In addition, atrophic changes in the brain and dura might constitute favorable conditions for circulatory disturbances in the granulation tissue that forms as the result of an exudate of blood or fibrin. In short, he believed that subdural membranes were derived from the organization of an inflammatory exudate, and owed their peculiarities of structure to the mechanical relations of the normal meninges, and to changes in them caused by systemic disease.

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27. Melnikow-Raswedenkow, N.: Histologische Untersuchungen über den normalen Bau der Dura mater, und über Pachymeningitis interna, Beitr. z. path. Anat. u. allg. Path. **28**:217-254, 1900.

Subsequent researches with elastic tissue stains, as in the cases of Jores and Laurent,[28] in a series of nonpachymeningitis duras examined in the course of the present investigation [29] and by other histologists [30] have not confirmed the constancy of the membrana elastica interna which Melnikow-Raswedenkow emphasized.

Jores [31] and his students Laurent [32] and van Vleuten [26] were engaged at about the same time in comparing cases of spontaneous pachymeningitis with those in which a subdural hemorrhage resulted from trauma. In the latter instance, and in the experiments on animals mentioned above, van Vleuten found that the hemorrhage was encapsulated and organized in the ordinary way by very vascular connective tissue. It then regressed and became a scar. These investigators, therefore, felt justified in stating that there was a traumatic, regressive form of pachymeningitis as well as a spontaneous, progressive form. The spontaneous form was characterized histologically in Laurent's cases by extreme vascularity of the membrane, often with enlargement of vessels and pigmentation elsewhere in the dura. In this form little connective tissue was seen. This is shown strikingly in one of the illustrations in his thesis. Feeling that the explanations so far considered were inadequate, Jores revived an old supposition advanced rather casually by Heschl,[13] namely, that spontaneous pachymeningitis is a neoplastic disease, a primary proliferation of the subendothelial layer of capillaries, sometimes complicated by subsequent hemorrhage, or exudation of fibrin and typical organization. This supposition has since been supported by Rössle [33] and Fahr.[34] In addition to these two forms, a variety caused by local infection was recognized by Jores, in which an inflammatory exudate was organized, with subsequent hemorrhages; but he does little more than mention the possibility of its existence.

Jores and his followers apparently had no opportunity to study any typical progressive or delayed traumatic cases, several of which had

28. Jores, L., and Laurent, H.: Zur Histologie und Histogenese der Pachymeningitis haemorrhagica interna, Beitr. z. path. Anat. u. z. allg. Path. **29**:486-506, 1901.

29. Putnam, T. J., and Putnam, I. K.: Experimental Studies on Pachymeningitis Haemorrhagica, to be published.

30. Nose, S.: Zur Struktur der Dura mater cerebri des Menschen, Arb. a. d. neurol. Inst. a. d. Wien. Univ. (Obersteiner) **8**:67-87, 1902.

31. Jores, L.: Ueber die Beziehungen primärer subduraler Blutungen zur Pachymeningitis haemorrhagica interna, Verhandl. d. deutsch. path. Gesellsch. **1**:49, 1898.

32. Jores and Laurent (Footnote 28). Laurent, H.: Zur Histogenese der Pachymeningitis haemorrhagica interna, Inaugural Dissertation, Bonn, 1898.

33. Rössle, R.: Zur Systematik der Pachymeningitiden, Centralbl. f. allg. Path. u. path. Anat. **20**:1043-1045, 1909.

34. Fahr., T.: Histologische Beiträge zur Frage der Pachymeningitis, Centrabl. f. allg. Path. u. path. **23**:977-981, 1912.

already appeared, mostly in the surgical literature. In such cases as that cited at the beginning of this paper, the vascularity may appear more marked than in certain spontaneous cases as, for example, Case 8 in Laurent's dissertation, and in the illustrations of Melnikow-Raswedenkow [27] and Cornil and Ranvier.[35] Moreover, there is reason for believing that the large blood filled spaces seen in some cases of hematoma of the dura are not strictly blood vessels. The vascular tissue has little resemblance to angiomas seen elsewhere. But it does seem to be true that one can recognize an extremely vascular and a comparatively nonvascular type of membrane.

In Great Britain independent investigations were being made by a number of observers, among whom Wiglesworth,[36] G. M. Robertson,[37] Ford Robertson [38] and Barrett [18] may be mentioned. Their papers are of particular value because they were written from a clinical as well as a pathologic point of view. Wiglesworth added little to what we have already learned of the pathologic anatomy of the condition, but laid emphasis on the frequency with which fresh fluid blood without a membrane was found beneath the dura in the insane. This occurred in one sixth of the cases that he reported. He argued from this fact that the membrane could not be the only cause of the hemorrhage, and that the reverse was probably true. He pointed out also that a general disease would be expected to be more widely spread over the dura, while subdural membranes were often unilateral and confined to the parietal region. He aptly compared the histologic picture to that of an organizing thrombus, and believed that the appearance of lamination need not imply repeated hemorrhages. He explained the hemorrhage by the rupture of a pial vein into the subdural space.

G. M. Robertson [37] published a careful survey of the literature and a clinical and pathologic study of the condition as seen in asylum material. He formulated what he called the "dry-cupping" theory, deriving the name from certain experiments in which Laborde produced congestion, ecchymoses and exudation in the serous surface of a rabbit's diaphragm by suction. Robertson believed that relatively negative pressure might occur in the surface of the dura as a result of discrepancies between the cerebral and the systemic blood pressures, and that engorgement and rupture of the delicate subendothelial capillaries might then result. He pointed out that "congestive attacks" are frequent in patients with paresis

35. Cornil, V., and Ranvier, L.: Manuel d'histologie pathologique, Ed. 3, Paris, **3**:10, 1907.

36. Wiglesworth, J.: Remarks on the Pathology of the So-Called Pachymeningitis Interna Haemorrhagica, Brain **15**:431-436, 1892.

37. Robertson, G. M.: The Formation of Subdural Membranes, or Pachymeningitis Haemorrhagica, J. Mental. Sc. **39**:203; 368, 1893.

38. Robertson, Ford: Pathology of Mental Disease, Edinburgh, 1900, p. 81.

and that a hyperemia of the dura is often found in alcoholic patients—the two groups of patients that furnish most of the cases of pachymeningitis. He mentioned atrophy of the brain as a supplementary cause of alterations in blood pressure, but did not suppose, as his terms might imply, that decrease in the amount of nervous tissue could cause a negative pressure on the dural vessels. Nontraumatic hemorrhage from vessels, if it occurs, must doubtless be due either to disease of the vessel wall or to variations in pressure within or without the vessels; but the character of the normal subendothelial capillary system is not such as to suggest the probability of rupture from ordinary pressure changes alone. Moreover, a case is reported in this paper (Case 9) in which a cyst developed in the face of intracranial pressure.

In a volume that has never received the attention it deserves, Ford Robertson [38] devoted most of one chapter to a consideration of the pathology of subdural membranes from a broad point of view. He was able to supplement G. M. Robertson's work with numerous careful histologic observations of his own, using especially an ingenious method for obtaining superficial horizontal sections. By these means, he was able to demonstrate certain changes that he believed preceded actual membrane formation in the insane. These changes were, grossly, the appearance of minute granulations of rusty straining or of a silvery, frosted sheen on the surface of the dura, and, microscopically, the proliferation and degeneration of the endothelial lining of the perivascular channels, which he demonstrated about the subendothelial capillaries, with occlusion of the vessels, diapedesis and rupture. At the same time the capillaries proliferated and grew large. Small quantities of blood or fibrin were thus extravasated and became organized in the usual manner. These hemorrhages were multiple; occurring from many capillaries at once, and the fibrin formed by subsequent hemorrhages might apparently surround and choke off capillaries already formed; that is, the membranes, if not renewed, had a tendency to regression, as Virchow had already suggested. The author produced clinical evidence to indicate that the membrane might be formed as an agonal event, and that organization might begin within a few hours. He agreed that a traumatic effusion of blood from rupture of a pial vein might give rise to a precisely similar process, and cited three original cases in which this was demonstrated.

Ford Robertson's chapter on subdural membranes remains the most thorough, original and adequately illustrated histologic account of them that we possess. The material on which the present paper is founded does not permit a complete repetition of his work, but it is confirmatory as far as it goes, and there is nothing in the histologic descriptions given by other authors to contradict what Robertson has described. His account of the blood pressure changes which he supposed to occur behind

an obstruction, and of the mechanism of obliteration of capillaries, is perhaps untenable, and he gave no satisfactory explanation of the occurrence of continued or late symptoms after a traumatic hemorrhage, but his paper is nevertheless a most valuable one.

Barrett,[18] in 1902, called attention to the fact that a fibrin network was often seen within the large, thin walled spaces containing blood which were found constantly in recent and progressive cases. Sometimes the fibrin within the vessel communicated with that outside. He believed that this intravascular clot caused obstruction and eventually rupture of the vessel, as had been postulated by Ford Robertson from obstruction by cellular proliferation, and by Melnikow-Raswedenkow from obstruction by the internal elastic membrane. Such an explanation is scarcely tenable from a consideration of the hydrodynamics involved; the vessels that are tied during an operation do not rupture, nor do capillaries that are obstructed by miliary emboli. Another explanation of the presence of a fibrin network is suggested below. The most important section of Barrett's paper is that which deals with the bacteriology of the subdural membranes. Bacteria were absent from smears, sections and cultures, and animal inoculation was negative. The author concluded that it was merely a forensic question whether the fibrinous exudate that initiated the change of pachymeningitis should be considered inflammatory or not.

Attempts have been made from time to time to prove that a local infectious process is responsible for the changes seen in pachymeningitis hemorrhagica. In some cases, definite purulent abscesses have been found in the membrane, as by Gläser [39] and Kluck,[40] and infections elsewhere in the body, even in the leptomeninges are not uncommon. Under such circumstances it is not surprising that bacteria should be found in the dura also, as was done by Roth [41] and Schwartz.[42] But cultures were constantly negative in the hands of Barrett,[18] Rosenberg [43] and others, and fresh tissue from the specimens examined for this review, stained with methylene blue, showed no bacteria. Büdinger [44] called attention to the nature of the fluid contained in hemorrhagic cysts; he thought it

39. Gläser: Vereitertes Hämaton der Dura mater, Deutsch. med. Wchnschr. **12**:815, 1886.

40. Kluck, M.: Ein Fall von vereitertem Haematoma durae matris, Inaugural Dissertation, Greifswald, 1890.

41. Roth, W.: Zur Genese und Aetiologie der Pachymeningitis haemorrhagica interna, Berl. klin. Wchnschr. **57**:175-178 (Feb. 23) 1920.

42. Schwartz, A. B.: Etiology of Pachymeningitis Haemorrhagica Interna in Infants, Am. J. Dis. Child. **11**:23-32 (Jan.) 1916.

43. Rosenberg, O.: Die Pachymeningitis haemorrhagica interna im Kindesalter, Ergebn. d. inn. Med. u. Kinderh. **20**:549-638, 1921.

44. Büdinger, K.: Die Ursachen der Spätsymptome nach duralen Blutungen, Med. Klin. **17**:584-588 (May 15) 1921.

resembled the contents of an infected hematoma rather than simply old blood. His cultures were sterile, however. Certainly local infections of the dura may occur, either by pyogenic organisms, by tuberculosis (Laurent [45]) or by syphilis (Hassin,[46] Wohlwill [47]), but the picture that such cases present is not likely to be confused with that of the group of cases under discussion, nor are the symptoms similar.

Pachymeningitis is not uncommon in children, occurring in 17.8 per cent. of necropsies on children up to 10 years of age, and 27.2 per cent. up to 1 year (Doehle [48] and Weihe). Others, among them Huegenin, give far lower figures. It may occur during the course of infectious or wasting diseases, and the onset may be acute or gradual. In a comprehensive paper, Rosenberg [43] has recently reached the conclusion that the condition is a result of venous obstruction; but other views have been strongly defended.

Traumatic Hematoma of Dura.—In the literature so far reviewed, only passing attention has been paid to cases of hematoma of the dura definitely resulting from trauma. Thibert, in 1822, encountered a probable case in which the onset of symptoms was delayed nine years after the trauma; and similar cases (Schuberg [6]) with longer and shorter intervals have appeared in almost every comprehensive article on the subject. Henschen [49] was able to compile 246 cases of subdural hemorrhage, some with immediate, some with delayed symptoms, and Brion [50] and Bowen [51] summarize more than eighty of them. The imposing collection compiled by Kasemeyer [22] established the fact beyond a doubt of the not infrequent occurrence of cases with late onset of symptoms, and his material has been drawn on freely in the present discussion. He reviewed at length about sixty case reports, only two or three of which are open to question, and yet he omitted practically all the cases appearing in the surgical literature, which are referred to in the other compilations just cited. Kasemeyer emphasized the medicolegal importance of these cases, which are often regarded as traumatic neuroses or psychoses of

45. Laurent (Footnote 32, second reference).

46. Hassin, G. B.: Histogenesis and Pathology of Subdural Hemorrhages, M. Rec. **44**:669-673 (Oct. 19) 1918.

47. Wohlwill: Ueber Pachymeningitis haemorrhagica interna, Arch. f. path. Anat. **214**:388-408, 1913.

48. Doehle: Ueber chronische Pachymeningitis bei Kindern und deren forensiche Bedeutung, Verhandl. d. X. Cong. f. inn. Med., Section 17, **5**:40, 1890.

49. Henschen, K.: Diagnostik und Operation der traumatischen Subduralblutung, Arch. f. klin. Chir. **99**:67-107, 1912.

50. Brion, W.: Die operative Behandlung der intraduralen Blutungen traumatischen Ursprungs, Inaugural Dissertation, Strassburg, 1896.

51. Bowen, W. H.: Traumatic Subdural Hemorrhage, Guy's Hosp. Rep. **59**:21-154, 1905.

other sorts; this was also mentioned by Nonne [52] and Homburger.[53] His discussion was mainly clinical, but he inclined to the opinion that merely the organization of a traumatic hematoma may give rise to repeated hemorrhages from rupture of capillaries.

PATHOLOGY OF SUBDURAL HEMATOMAS

Bearing in mind the various theories that have been suggested to account for this disease, and the points that have been brought forward to support them, we may proceed to a description and analysis of the appearances seen in the clot, its membranes, the dura, and the remainder of the body in such cases. The appearances found vary from case to case, but in general we may recognize an essentially vascular type, and one that might be termed a reactive type of membrane. The former (pachymeningitis vasculosa of Orth) has been frequently described, since the time of Virchow, in patients suffering from chronic wasting diseases and without a history of trauma. In the latter type, with which the present paper is chiefly concerned, a definite history of trauma has usually been obtained, although sometimes not until after the lesion has been demonstrated by operation or necropsy. The lesion has therefore been called pachymeningitis traumatica, but it is distinguished from the spontaneous form by histologic as well as by clinical peculiarities, at least in the cases here reported and in those adequately described or illustrated in the literature. Apparently a similar condition may be seen in certain hemorrhagic diseases. The gross appearance of the two forms is practically identical, and the microscopic pictures are so similar that they are easily confused. The symptoms may also be similar, except that in the nontraumatic form they are usually masked by the manifestations of the primary disease, and are less likely to lead to an operation, though some cases have been operated on (Starr,[54] Jaboulay [55]). Through the kindness of Dr. Canavan of the Warren Museum, Dr. Fulstow of the Boston Psychopathic Hospital, Dr. Mallory of the Boston City Hospital and Dr. Wolbach of this clinic, it was made possible for us to compare the slides of six spontaneous cases of pachymeningitis with the specimens from the surgically treated cases reported here, and also with those described or illustrated elsewhere. They correspond with the latter closely.

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52. Nonne, M.: Zwei Fälle von Hämatom der Dura mater, die mittelst Schädelborung diagnostiziert worden, München. med. Wchnschr. **54**:909-910, 1907.

53. Homburger: Ueber Pachymeningitis haemorrhagica mit Korsakowscher Psychose und atypischer Hemiplegie nach Trauma, München. med. Wchnschr. **52**:2393, 1905.

54. Starr, M. A.: Organic Nervous Diseases, New York, 1903, p. 709.

55. Jaboulay, M.: Hématome de la dure-mère, Arch. prov. de chir. **2**:61, 1893.

As the appearances seen in the clot itself, the membrane above, the dura, and the remainder of the body are similar in the two forms, the two will be discussed together under those headings. The essential histologic difference between the traumatic or reactive and the spontaneous forms lies in the structure of the subdural portion of the sac enclosing the clot, and this subject will be discussed separately for the two varieties.

Location.—The process is bilateral in from one third to one half of all cases.[24] This may have an especial surgical significance.[56] Multiple cysts or hematomas on the same side have not been reported. The parietal region is the most frequent location, but membranes are also seen elsewhere. In Case 11, contributed by Dr. Naffziger, the hematoma was chiefly basal, and Ciarla has reported a case that was perhaps similar. Neisser and Pollack [57] mentioned two cases in which the process was in the posterior fossa. The pachymengitic process involved the spinal cord also in several cases in the literature.[2] Membranes alone may occur in two or more discrete patches on one side of the dura (Ciarla). The characteristic distribution has been cited both for and against the inflammatory theory of origin; on the one hand, because one would expect an inflammatory process to be more widespread; on the other hand, because birth hemorrhages in children are likely to lie on the tentorium.[43]

Size.—The cyst or hematoma may vary from that of a microscopic ecchymosis to a collection of fluid displacing half the substance of one hemisphere. Small ones are far more common than large, and it is important to remember that the subdural membrane reaches a thickness of possible clinical significance in only a fraction of the reported cases—in twenty-six of Ciarla's [24] 163 cases, and in four of Robertson's seventy-one cases, with recent large unencapsulated hemorrhages in eight.[38] Robertson states that the usual thickness is one-eighth inch (3 mm.) or less. Many subdural membranes contain no extravasated blood (Jores,[28] Melnikow [27]). Bowlby [58] speaks of finding a cyst as large as one's fist at necropsy in a man who had had no symptoms from it whatsoever. On the other hand, Starr [54] reports a case in which a child, aged 9, was operated on by McCosh six months after trauma to the head, which had been followed first by hemiplegia, then by convulsions. A thin, velvety tissue was curetted from the inner side of the dura; no other injury was disclosed. The child made a complete recovery.

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56. Trotter, W.: Chronic Subdural Hemorrhage of Traumatic Origin, and Its Relation to Pachymeningitis Haemorrhagica Interna, Brit. J. Surg. **2**:271, 1914.

57. Neisser, E., and Pollack, K.: Die Hirnpunktion, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **13**:807-896, 1902.

58. Bowlby, K.: Surgical Pathology, Ed. 3, London, 1895, p. 598.

The contents of the hematoma may vary from a fresh, reddish brown, gelatinous clot to colorless fluid. Usually a coffee colored fluid with clots is mentioned in cases coming to operation. Complete organization of a large clot has not been reported. The condition of the clot appears to have little relation to the length of the interval between trauma or onset of symptoms and operation or necropsy. Thus, in Cant's patient, operated on forty-five days after being kicked by a horse, a quantity of yellow fluid and a small clot was found.[59] The man recovered from the convulsions which began six days after the accident. On the other hand, in Case 1 of Buss'[21] article, there was found at necropsy on a man who died three and a half years after trauma to the head and a year and a half after the onset of symptoms, a small, fresh hematoma on one side and an old one the size of a fist on the other. There may be great variation in the contents of a single hematoma. In contiguous areas, like states on a map, will be a mass of organized tissue; next to it well preserved red cells with no fibrin; next, a heavy fibrin mesh; next, a collection of serum or hemolyzed cells. Lakes of fluid or of fibrin-free cells seem likely to become encapsulated rather than replaced by organization tissue. This is especially well shown in animal experiments. In one case (Case 3 of this series), a small excess of polymorphonuclear leukocytes was seen in proportion to the red cells. Usually they are scanty. Large wandering cells are sometimes to be found in situations that suggest that they are the forerunners of organization.

In most of the published accounts of the microscopic structure of hematomas of the dura, the occurrence of successive layers of different ages is described. In the cases observed during the present investigation (including the spontaneous cases and the experiments), the appearance of the clot varied in different parts of the same slide, as has been mentioned above, but not regularly in layers, and entirely without regard to the proximity of blood vessels. Moreover, the microscopic similarity between the certainly regressive hematomas produced experimentally and those occurring in human beings is against the assumption of repeated hemorrhages. Slow, gradual, continuous oozing might produce the appearances found. The question whether repeated hemorrhages occur is an open one. Lamination in a hematoma formed by a single hemorrhage is reported by Wiglesworth.[36]

In the cases with which we are particularly concerned, the clot is completely enclosed in an envelop of fibrous tissue. Over the surface toward the arachnoid, the membrane consists of a thin sheet of connective tissue covered with a single layer of mesothelial cells. The presence of blood vessels has been reported in this membrane, which is constantly

59. Cant: Case of Traumatic Epilepsy; Trephining; Recovery, Lancet **1**:542, 1891.

observed; but in the cases in the present series, and in most of those published elsewhere, it is less than a millimeter in thickness and avascular. It is usually free from the subjacent arachnoid. Occasional small synechiae are found, which in our cases have been bloodless. Virchow [16] believed that vascular connections could occur.

Subdural Membrane.—Unlike the tissue on the cerebral aspect of the hematoma, that on the dural side has a definite structure, which is of importance because this membrane may apparently give rise to hemorrhage under certain conditions. It will be necessary, therefore, to give a detailed account of it, and to describe the various appearances seen in different forms of hematoma of the dura separately.

Reactive or Traumatic Type of Subdural Membrane.—It seems probable that membranes of the type seen underlying traumatic hematomas may occur without the hematoma, but none have been reported, except the scarlike thickenings seen after operation or trauma described below, which usually lack certain of their distinctive features (van Vleuten,[26] Böckmann [60]). In the cases here reported, the layer between clot and dura is from 1 to 4 mm. in thickness. It is composed of well formed, fibrous tissue, the cells of which are numerous and distinct and have plump, oval nuclei. It is sharply marked off from the dura in most cases, for the fibers of the latter are slightly hyaline, and stain more deeply with eosin. In gross the junction between the two is found to be easily separated. It is less clearly marked off from the blood clot into which it sends lines and columns of fibroblasts and a few fine vessels. However, the surface of the vascular portion of the tissue is usually fairly uniform. The fibrous tissue contains wandering cells, pigment and occasional red cells and leukocytes in its meshes, especially about the vessels. In only two cases (Cases 5 and 11) were definite interstitial hemorrhages seen, and in one of them they may have been due to the trauma of surgical removal. Interstitial hemorrhages are mentioned by Virchow, Ford Robertson and others, but without satisfactory description. Laurent [45] observed interstitial hemorrhages in a case of purpura.

The interest in subdural membranes centers on the vessels and blood filled spaces. Typical capillaries are found, sometimes numerous (Fig. 3) and sometimes scanty (Fig. 4), with normal appearing structure and contents. Fibrin is not seen in undoubted capillaries. They may be somewhat larger than normal, but seldom strikingly so. As far as the fibrous tissue and vessels go, the membrane may be indistinguishable from exuberant granulation tissue seen elsewhere.

The characteristic feature of the membrane is the presence of large spaces of irregular form but in general elongated, most of which lie in

60. Böckmann: Ein Beitrag zur Aetiologie der Pachymeningitis haemorrhagica interna, Arch. f. path. Anat. **214**:380-388, 1913.

a definite, uniform line parallel to the surface of the dura, separated from it by the thickness of a few fibroblasts (Figs. 4 and 5). These spaces have a definite mesothelial lining, which, however, is sometimes incomplete and less uniform than the endothelium of the neighboring

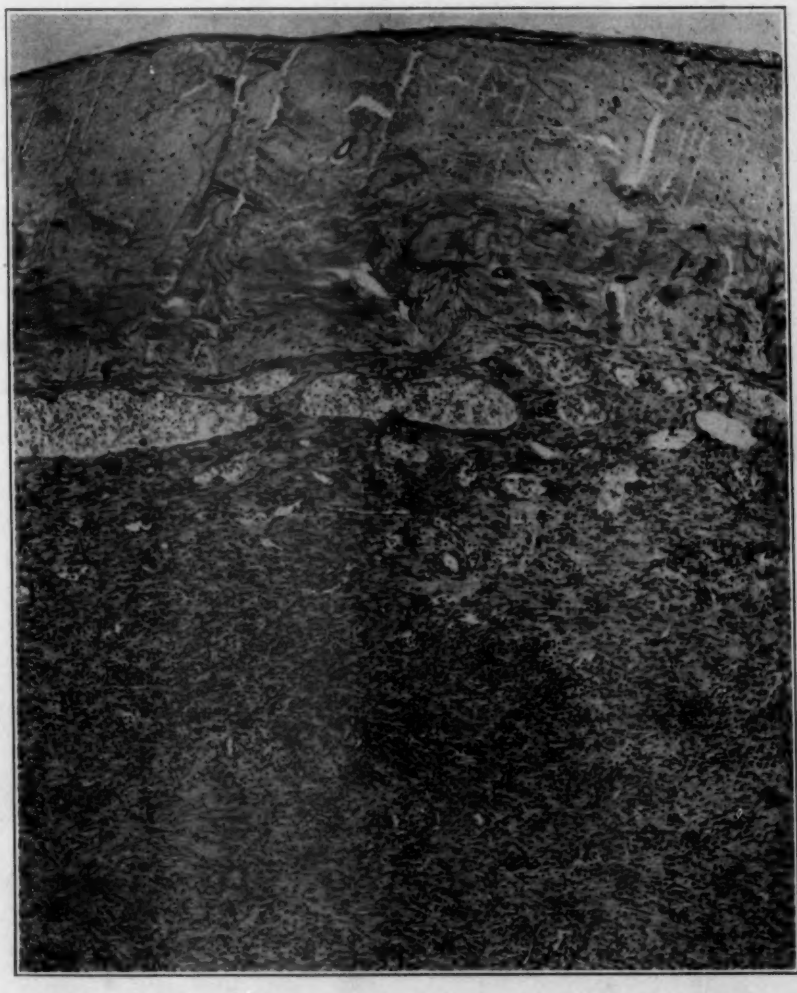

Fig. 4 (Case 3).—Membrane attached to dura, removed at operation; pale, hyaline, fibrous dura at top; its juncture with the darker, more cellular granulation tissue may be made out. Just below the line of cleavage is a row of large rounded spaces containing a little blood. The membrane beyond them is not very vascular. The clot lies outside of the picture. An unusual number of polymorphonuclear leukocytes is present. Methylene blue and eosin stain; x 80.

capillaries. The cells are sometimes plump, sometimes flat, and the intervals between the nuclei vary. A few of the spaces may appear practically empty, but usually they contain red cells, with occasional

leukocytes, fibrin or granular débris. They seldom give the impression of being stuffed with blood, as the capillaries often do, but the cells within them are perfectly preserved, and seem to have no tendency to become absorbed. Occasionally the fibrin that they contain appears to communicate with the network outside, as described by Barrett.[18] Spaces were absent from only one of the seven cases examined.

These spaces occasionally have a rounded or serpentine outline, which is sometimes so regular as to suggest that they form a continuous series of tubes. From vertical sections, it is difficult to say whether they communicate with each other and with the neighboring capillaries, although

Fig. 5 (Case 2).—Thin portion of membrane stripped from dura at operation. The enormous size and fantastic shape of the mesothelium lined spaces lying just below the dural attachment is well shown. One of them appears to lead into a capillary. The tissue is otherwise not very vascular. Little is left of the contents of the hematoma; the clot at the bottom of the picture was probably formed at the time of operation. Methylene blue and eosin stain; x 80.

they apparently do so (Figs. 5 and 6). In sections cut parallel to the plane of the dura, their irregular anastomoses and their connections with small vessels of the dura and capillaries of the organization tissue are well shown (Fig. 7). In general, it appears that the longer the interval between the trauma and the removal of the specimen, the larger and more irregular are the spaces. The fact that the spaces described are removed intact when the membrane is stripped from the dura, instead of

forming a line of cleavage in themselves, is evidence that their walls have a definite strength and structure and that they are not artefacts. Von Saar and Herschmann [61] appear to be the only authors who have observed similar spaces.

Structure of Subdural Membrane in Experimental and in Post-operative Hematoma of Dura.—Small, regressing, subdural hematomas may sometimes be found in patients dying after intracranial operations, and may be produced experimentally in animals. Such hematomas become covered within a week by a thin layer of fibrous tissue bearing

Fig. 6 (Case 2).—Section from the same slide as Figure 5. The character of the lining of the large blood filled space at the top of the picture may be compared with that of the capillaries below. A communication between them is suggested. A thick strand of fibrin runs into the blood filled space. Methylene blue and eosin stain; x 300.

mesothelium. They become organized from the dura by granulation tissue which contains large spaces lined with mesothelium (Fig. 8) much like those discussed above. But the spaces are practically always empty, and do not seem to open into definite blood vessels. Such spaces are rarely seen in organization tissue in other locations (that replacing epidural hematomas, for example). It is probable that their origin or persistence depends on the pressure relations within the skull, or on the peculiarities

61. Von Saar and Herschmann: Zur Symptomatologie und Therapie der Pachymeningitis haemorrhagica interna, Deutsch. Ztschr. f. Chir. **145**:398-414, 1918.

of the dural mesothelium, or both. The granulation tissue is usually less vascular, perhaps because the clot is thinner than in the cases just described. Yet, though it is thicker than some of the subdural membranes that occur without trauma, it fails to show their characteristic plexus of vessels. We are unfortunate in having no complete account of the process of organization and the fate of simple hematomas of the dura of a size comparable to those reported in the present series. Perhaps Bowlby's [58] was such a case. It does not seem probable that they are all progressive.

Cleared specimens and horizontal sections after a capillary injection with India ink in animal experiments show that the lined spaces are discrete and do not communicate with blood vessels.

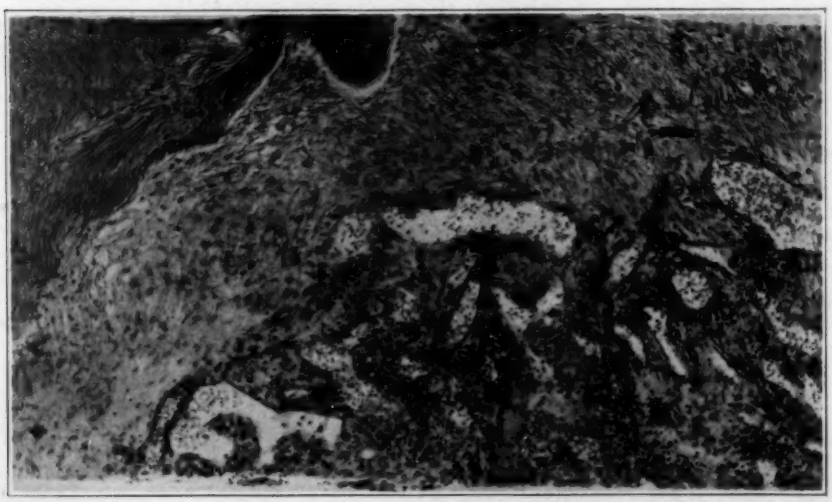

Fig. 7 (Case 2).—Section from same block of tissue as Figures 5 and 6 but cut in a plane parallel to the dura through many blood filled spaces. Their varied forms and their anastomoses with each other and with capillaries are well shown. Some of them contain fibrin. Hematoxylin and eosin stain; x 80.

Structure of Membrane in Nontraumatic Hematoma of Dura.—Similar changes were seen in all six of the cases of spontaneous pachymeningitis at our disposal, and they agreed in general with the descriptions given by Virchow, Jores, Laurent and Ford Robertson. The appearances are most beautifully shown in two of the three sets of slides loaned by Dr. Mallory, one of which is illustrated (Figs. 9 and 10). The presence of many blood vessels may be evident to the unaided eye. Microscopically, it is seen that they have the structure of capillaries, although they may reach a diameter of half a millimeter. In the specimen illustrated, they are stuffed with blood. In other cases, they may

be collapsed, so that one gets the impression of a tissue less vascular than, for example, that shown in Fig. 7. This was observed by Ford Robertson, who believed the condition of the capillaries to depend on the mode of death.

The vessels vary considerably in size, but it is unusual to find any of them smaller than the largest capillaries seen in membranes of the

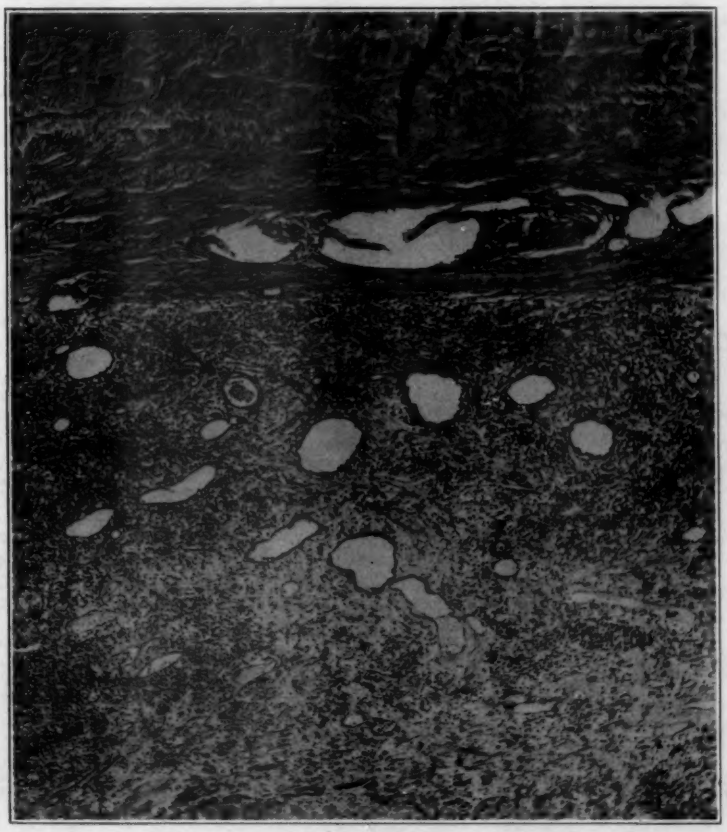

Fig. 8.—Specimen from the dura of a patient who died of pneumonia a month after an exploratory operation for brain tumor. There was some intracranial pressure. The firm organization tissue springing from the dura contained many large spaces, evidently anastomosing, but empty. Blood vessels are rare. One may be seen, full of blood, in the center of the field indicated by the arrow. Hematoxylin and eosin stain; x 80.

traumatic type. The largest of the "giant capillaries" usually lie in a row a little above the plane of separation from the dura, as to the mesothelium lined spaces. The spaces, however, are often larger, more varied and more fantastic in form than the vessels. The latter usually

have a rounded outline, as if they were all a part of a continuous network. That such is the case is indicated by horizontal sections like those of Ford Robertson,[38] or by teased preparations (Fig. 11) like those shown in Barrett's [18] paper and in Laurent's thesis.[45] The continuity of the largest vessels that lie in a line close to the dura has not been demonstrated, but they probably anastomose much like the blood filled spaces. Communications between a large vessel and a small one are seldom seen, but do occur. In the cases at our disposal, no degen-

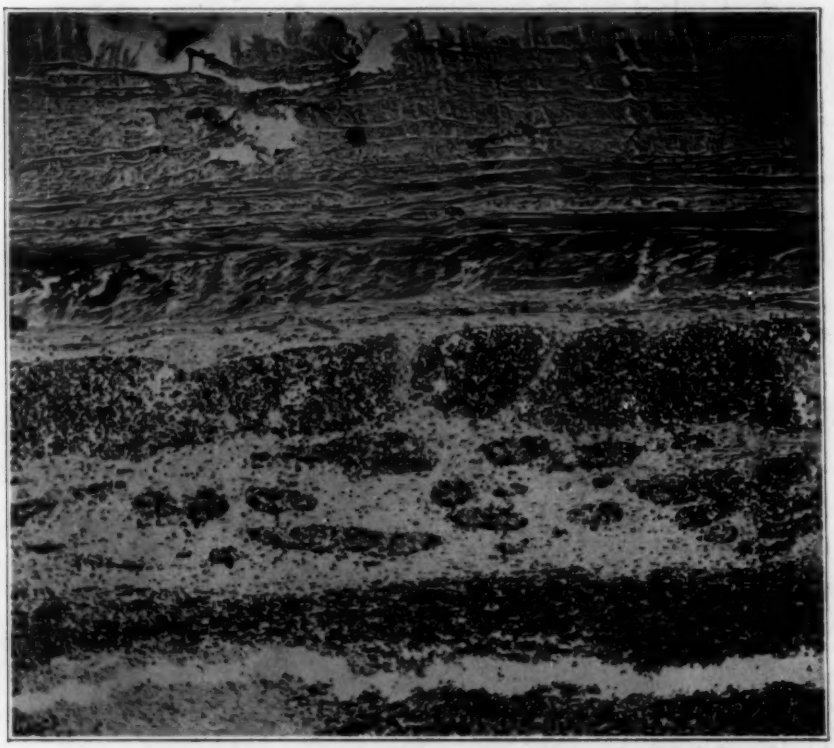

Fig. 9.—Necropsy specimen of spontaneous pachymeningitis, loaned by Dr. Mallory; hyaline dura at the top. The membrane is filled with a plexus of "giant capillaries," apparently communicating with the large spaces stuffed with blood below the dural surface. The edge of a large hematoma is shown, but there appears to be little tendency to organization. Methylene blue and eosin stain; x 80.

erative changes were seen in these vessels. Lined spaces containing serum, débris or fibrin are rarely found in the spontaneous type of membranes. When they exist they are usually small.

The stroma in which the vessels lie is firm and fibrous, and usually gives the impression of being older than in the traumatic cases. It

contains pigment, but few leukocytes. It is sharply marked off from the hyaline dura. The boundary between it and the clot is sharper than in the cases previously described, and organization is carried on more indolently, with fewer vessels. In only one of the specimens at our disposal were there interstitial hemorrhages. The only other

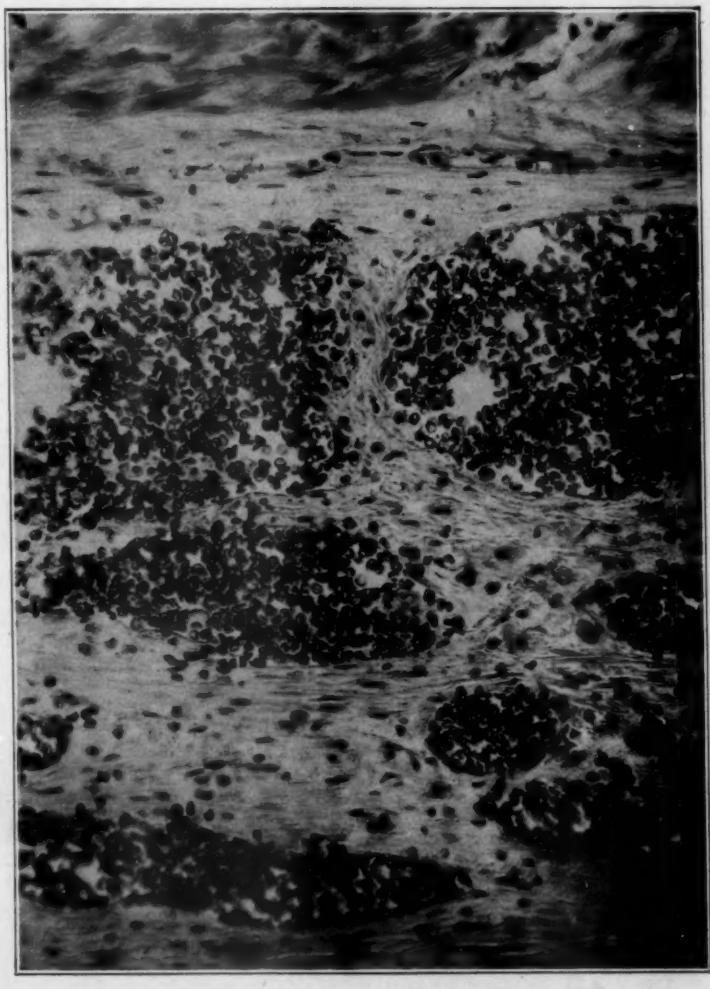

Fig. 10.—Section from the same slide as Figure 9. The "giant capillaries" are seen to have only an endothelial covering. The firm, uniform character of the stroma is shown. A definite boundary between it and the hyaline dura may be seen at the top of the picture. Methylene blue and eosin stain; x 300.

definite evidence of successive layers of organization was the presence of a layer of fibrous tissue containing normal sized capillaries, between the vascular membrane and the hematoma.

Spontaneous subdural membranes are most frequently seen without hematomas, unlike the traumatic variety. Such membranes may be more vascular than the ones in which hemorrhage has occurred.[45] None were studied in this series, and the reader may be referred to Ford Robertson's paper [38] for a discussion of them.

Regressive Changes in Subdural Membrane.—It is evident from an examination of a series of subdural membranes of various origins that their tissues are subject to degenerative changes like any others. A hyaline appearance is common in older membranes. Calcification was

Fig. 11.—Horizontal teased preparation, from the dura of a brain loaned by Dr. Fulstow of the Boston Psychopathic Hospital. The patient was demented. At necropsy a bilateral hemorrhagic dural cyst was found. Vertical sections resembled those shown in Figures 9 and 10. In horizontal preparations a coarse plexus of large vessels is seen. Hematoxylin and eosin stain; x 80.

not observed in the present series, but has been described (Lewis [62] and Elsner [63]). Nor did fatty degeneration of the vessels of the membrane itself occur, as mentioned by Charcot and Vulpian.[7] Ford Robertson

62. Lewis, B.: Textbook of Mental Disease, London, 1889, p. 434.

63. Elsner, K.: Ueber Pachymeningitis Ossificans, Inaugural Dissertation, Munich, 1896.

gives a precise account of various morbid products that are seen in thin membranes and the subjacent dura. Purulent infection of the hematoma itself has been alluded to above. It did not occur in a case that Dr. Canavan of the Warren Museum kindly allowed us to examine, in which a hematoma of the dura supervened on a purulent leptomeningitis in a bedridden old woman.

Changes Seen in Dura.—In general, the dura is little altered. It appeared slightly hyaline in most of the cases of this series, but so it does in many otherwise normal specimens. The normal lacunae of the substance of the dura seem to play no part in the morbid process, and it is perhaps not out of place to remark here that fewer of them are seen in specimens taken from the convex surface than one would be led to suppose by the usual descriptions. The small interstitial hemorrhages observed by Nose [30] in his study of normal duras were seen in one specimen. Granules of pigment are frequently seen even in the depth of the tissue. Variations in the thickness of the dura itself are inconstant and probably of little significance, unless extreme. An increase in the thickness and vascularity of the subendothelial layer has been described by Melnikow,[27] by Laurent,[45] and especially by Robertson [38] as an early change. A slight infiltration of the submesothelial zone with round cells, considered by Robertson to be endothelial, is frequently seen. Other leukocytes are rare.

Atheroma of the arteries of the dura occurred in several of the traumatic cases. Surprisingly enough, it was not seen in the spontaneous ones. It has been reported, however, by Charcot [7] and others. Ford Robertson rarely found atheroma of the intracranial vessels in either alcoholic or paretic patients. There appeared to be slight enlargement of the vessels of the dura, especially of the submesothelial plexus, in some cases, but the importance of this change is perhaps overemphasized by Jores and Laurent.

Changes in Pia and Arachnoid.—The leptomeninges are seen to be yellowed at operation or at necropsy. They were studied microscopically in only one of the surgical cases, and were found normal. In one of the spontaneous cases, there was considerable fibrosis of the arachnoid spaces. The pial vessels appeared normal. In another case there was a purulent meningitis. In the others no specimen of them was kept. A similar thickening of the arachnoid, with obliteration of some of its meshes and adhesions to the neomembrane was seen in experimental animals. Great importance was attached to this phase by Ramaer [64] and by Hassin,[46] who regarded the arachnoid as the primary seat of the disease and the pia as the source of the hemorrhage. In a case given

64. Ramaer: Bemerkung zur Abhandlung über das Hämatom der Dura mater von Dr. G. Weber, Arch. f. path. Anat. **24**:223-224, 1862.

by Bondurant,[65] a subarachnoid hemorrhage also was found, suggesting that both originated from a rupture of the same pial vein. The spinal fluid is frequently xanthochromic, sometimes bloody, sometimes normal.

Changes in Brain.—The deformity of the brain is striking, as is shown in illustrations published by Ribbert,[66] Sachs [67] and Heuer and Dandy.[1] Microscopic changes attributable to the hematoma have not been demonstrated in it, but are stated to have been produced experimentally.[37] Trotter [36] observed absence of pulsation of the brain and apparent thrombosis of the cortical vessels in two fatal cases. Of course, most cases occur in the insane. Atheroma of the arteries of the base is not infrequent.

Apoplectic foci are said to be among the local causes of pachymeningitis by Kremiansky,[23] who reports such a case. A few cases have been published in which the hematoma appeared to have originated from a cortical apoplectic focus (communicating apoplexy). In general, subdural membranes are seldom seen in patients dying of apoplexy.

Hemorrhage into the optic sheaths has been observed, especially in children (Zacher,[68], Fürstner,[69] Rosenberg [43]). Destruction of the eighth nerve was reported by Moss (Kasemeyer [70]).

Changes in Skull and Scalp.—Thinning, thickening and roughening of the surface of the skull were mentioned by various of the earlier writers,[20] but have been largely neglected by later ones. At operation the bone has sometimes been found thicker and more vascular than normal, but no other changes are mentioned. In three cases (those of van der Graaf,[71] of Heuer and Dandy,[1] and Case 10 observed by Naffziger), there have been changes in the cranium which were demonstrated by roentgen-ray examination.

No constant changes are found in the scalp. The tendency to postoperative hemorrhage is probably no more marked than in cases of other kinds. Hematoma of the ear has been observed in some of the

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65. Bondurant, E. D.: Pachymeningitis Interna Haemorrhagica, with Report of Eight Cases, Alienist & Neurol., St. Louis, **14**:1-11, 1893.

66. Ribbert, H.: Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie, Ed. 9, Leipzig, 1923, p. 419.

67. Sachs, Ernest: Eight Years' Experience with Brain Tumors, Arch. Surg. **1**:74 (July) 1920.

68. Zacher: Doppelseitige Stauungspapille mit Perineuritis bei Hämatom der Dura mater, Neurol. Centralbl. **2**:125-132, 1833.

69. Fürstner, C.: Zur Genese und Symptomatologie der Pachymeningitis haemorrhagica, Arch. f. Psychiat. **8**:1-30, 1878.

70. Kasemeyer, E.: Post-traumatische Pachymeningitis, Tod 5 Jahre nach dem Unfall, Deutsch. med. Wchnschr. **38**:2020, 1912.

71. Van der Graaf, A. M.: Die Roentgenphoto als Hulpmiddel bij het Herkennen van het Haematoma durae matris, Nederl. Tijdschr. v. Geneesk. **2**:1837-1840, 1915.

cases among the insane and is perhaps related pathologically to the condition under discussion.[38] It exists also in both a spontaneous and a traumatic form.

Changes Elsewhere in Body.—There are no constant somatic changes in traumatic hematoma of the dura. The frequency of lung complications has been stressed, but does not seem remarkable. Albuminuria is often seen, but structural renal lesions are unusual. The reader is referred to the Ciarla [24] and Huegenin [20] for a description of the multiform coincidental changes seen with the spontaneous variety.

ETIOLOGIC FACTORS

Traumatic Hematoma of Dura.—Aside from trauma, no single antecedent factor is common to all cases. The disease often occurs in young, healthy, temperate men. Chronic alcoholism has been thought to predispose to meningeal hemorrhage ever since the time of Lancereaux,[72] and addiction to the use of alcohol to some extent appears rather frequently in the histories of Bowen's [51] collection. In the present group of cases, studied for this article, four were known to be very temperate or abstainers, two were moderate drinkers, two regular or heavy drinkers, and in two cases the history was incomplete. Kasemeyer [70] attaches little importance to the occurrence of alcoholism, and it appeared seldom in the histories of Bowen's [51] compilation. In the present group of cases, no acute or chronic infectious disease occurs. Generalized arteriosclerosis was seen in one patient. Two patients were perhaps slightly paranoid, one epileptic, one hydrocephalic. Mental disease occurs in few of the cases reported elsewhere in surgical literature, but Ford Robertson in asylum material found some cases without a preceding pachymeningitis.

Type of Trauma.—In one case (Case 5) there was no history of trauma. The absence of other etiologic factors and the pathologic findings make it seem permissible to group it with the others. The trauma may be very slight, and a history of it may be obtained only after operation or necropsy, as in Cases 7 and 8, and in two of Trotter's six cases. Indeed, it is rather surprising that the history of trauma has been so frequently elicited in the published cases. Most of the cases in which it has not been obtained have probably been classified in the other group.

The trauma may be very slight. A football injury with only momentary giddiness and no disability occurred in Case 1. A slap in the face,[73] a fall from a step,[40] or a blow on the chin [51] may initiate a hemorrhage.

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72. Lancereaux: Des hémorrhagies meningées, considerées principalement dans leurs rapports avec les néomembranes de la dure-mère cranienne, Arch. gén. de méd. **2**:526-550; 679-696, 1862; ibid. **1**:38-67, 1863.

73. Hofmann, E.: Schläge mit der Hand in's Gesicht, Tod durch Pachymeningitis haemorrhagica, Wien. med. Presse. **17**:1437-1440, 1876.

Repeated slight trauma may produce the condition, as in the case of Trotter's in which the patient kept bumping his head against a low beam in the shop where he worked, and in five cases cited in Kasemeyer's article, in which the children had been subjected to habitual abuse by their guardians. More often, however, the violence is considerable, and the patient is unconscious for several hours or longer. Trotter suggests that frontal or occipital blows are more provocative than those in the parietal regions, because the brain is supported by the falx against lateral violence.

Age Incidence.—Hematoma of the dura is usually considered a disease of senescence, but in this series the patients' ages ranged from 17 to 58 years, half of them being less than 36. Cases are not rare in children.

Nontraumatic Hematoma of Dura.—Brief allusion may be made to the spontaneous form of pachymeningitis for comparison. More complete discussions will be found in the papers of Ford Robertson, Ciarla, Huegenin and Rosenberg. No cases have been reported without some precedent disease. Most occur in the insane, especially in paretic patients, but the condition may be found in persons dying from any common, wasting disease. The concurrence of chronic alcoholism has been mentioned. Bertholet [74] found pachymeningitis in 10 per cent. of necropsies on alcoholic patients, and it has been reported in about the same proportion of animals fed alcohol over long periods.[25] Diseases accompanied by violent coughing, such as tuberculosis and pertussis, and congestive heart disease have been considered of special importance. One tenth of Huegenin's cases accompanied pernicious anemia, and examples are seen also in other hemorrhagic diseases, such as scurvy [75] and purpura. In such cases the microscopic picture may resemble that of the traumatic type (Laurent,[45] Wohlwill [47]).

Does trauma ever produce a true vascular pachymeningitis? The possibility cannot be excluded, but the occurrence has never been absolutely demonstrated. Case 6 of Jores and Laurent's series [28] may represent an example. The subject will be discussed in a later section.

SYMPTOMATOLOGY

Interval Between Trauma and Onset of Symptoms.—Bowen [51] makes an important distinction between the "lucid interval" occurring before the onset of unconsciousness alone and the "latent interval" before the onset of symptoms of intracranial pressure or cortical irritation. The

74. Bertholet, E.: Petit atlas-manuel des altérations anatomio-patholgiques dans l'alcoolisme chronique, Lausanne, 1913.

75. Sutherland, G. A.: On Hematoma of the Dura Mater, Associated with Scurvy in Children, Brain **17**:27-36, 1894.

latter is likely to be longer than the former, and is characteristic of subdural hemorrhage. In his compilation, and those of Brion [50] and of Henschen [49] it is seen that the interval may be only a few hours, with death within a day; or symptoms may be delayed for months. In a case given by Buss,[21] a healthy, temperate man began to have vague cerebral symptoms two years after a severe fall, and at necropsy eighteen months later, a subdural hematoma was found. In Henschen's series of 121 cases of subdural hemorrhage, the latent interval in thirty was less than a day, in seventy-seven less than a week, and in 103 less than three weeks.

Delayed symptoms, therefore, are surprisingly common and we may turn our attention chiefly to their consideration, though aware that no hard and fast line can be drawn between the early and the delayed cases.

Two cases (Cases 1 and 11) are included in the present series in which slight symptoms began soon after the accident. Severe symptoms and objective signs, however, did not appear until two weeks later in one case, twenty months later in the other; in both the characteristic pathologic picture was revealed at operation. A few similar cases have already been reported, and throw an interesting light on the mechanism of the condition.

Duration of Symptoms.—In general the longer the latent interval, the more gradual is the onset of symptoms. Yet Wells' patient [76] fell in a convulsion on the street five weeks after an injury to his head, having had only slight headache and weakness for twenty days. Kasemeyer [70] reported a case in which the symptoms lasted five years before death. A week to several months is the usual time elapsing before the symptoms demand operation or become fatal.

General Symptomatology.—The symptoms of the traumatic type of hematoma are so like those described for the spontaneous type that no distinction will be made between them in the present discussion. Perhaps the reason that more of the former than of the latter are operated on is because the history of recent trauma may lead the patient into surgical hands.

It is customary, following Schuberg,[6] to attempt to divide the course of the disease into a period of incipiency and a period of disturbed motility, but this is often impossible. The symptoms show a gradual, most irregular progression. Indeed, an extraordinary variability is their most pronounced characteristic, as pointed out by Fürstner.[69] Trotter explains this lability by supposing that when the hematoma reaches a size that barely begins to produce symptoms, a physiologic diminution in the size of the brain may dispel them again, only to reappear later.

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76. Wells, F. L.: An Interesting Case of Subdural Intracranial Hemorrhage (Without Fracture); Leptomeningitis; Trephining; Recovery, M. Rec. **41**:541-542, 1892.

Very little more need be said about the general symptoms. They are amply illustrated by the appended histories. They may be divided into those due to general intracranial pressure (headache, vomiting, vertigo, slow pulse, choked disk); those due to local pressure (paralyses, rarely sensory disturbances, spasticity, convulsions, aphasia), and perhaps those due to irritation (Kernig's sign, stiff neck). They may occur in almost any combination, and besides being variable and fleeting, are apt to be atypical or incomplete, to an extent that is difficult to explain. Thus, a pure anomia may appear and reappear as a result of a lesion involving almost the entire hemisphere. Or a plantar reflex may be absent in the presence of a hemiplegia.[77] Of especial interest is the question of the occurrence of a homolateral hemiplegia which has been reported in this disease by four authors.[78] Sometimes paralytic or irritative symptoms are evoked from both hemispheres by a unilateral lesion, but more often a bilateral hematoma gives symptoms on one side only.

Special Symptoms—Certain clinical manifestations of hematoma of the dura are more or less characteristic, though often not satisfactorily explained by the pathologic findings.[79] The extreme variability of symptoms is in itself a diagnostic point of the greatest value. The patients are apt to be very unmanageable, on account of a certain irritability, restlessness and perversity, sometimes amounting to negativism. It may seem that they are wilfully untidy and obscene. Two of the patients seen at this clinic had to be provided with male nurses. Much emphasis has been laid on oculomotor abnormalities. There may be areflexia, anisocoria, miosis, conjugate deviation, ptosis, strabismus, etc., which have a diagnostic, but seldom a lateralizing, significance. Hemianopsia was reported by Ziehen and van der Scheer [80] and anosmia by Tuczek (Kasemeyer [22]). Deafness was found in two cases by Ciarla. Bárány tests were performed on two of the patients in the present series and were negative. There may be local tenderness and dulness on percussion may be recognized by simultaneous auscultation over the forehead.[80]

The temperature is usually slightly elevated. In the patients of von Saar and Herschmann,[61] aggravation of symptoms was each time accompanied by a rise in temperature. No precise relation between the two was seen in the cases observed here. Subnormal temperature, suggesting brain abscess, sometimes occurs. The pulse is low. This bradycardia may persist with excitement or activity. The blood pressure is

77. Homburger (Footnote 53). van der Scheer, W. M.: Zur Klinik der Hämaton subdurale, Ztschr. f. d. ges. Neurol. u. Psychiat. **23**:66, 1914.

78. Ciarla (Footnote 24). Ledderhose: Ueber collaterale Hemiplegie, Arch. f. klin. chir. **51**:316-349, 1895.

79. Griesinger, W.: Ein Heilungsfall, Arch. f. d. ges. Heilk. **3**:33-46, 1862.

80. Van der Scheer (Footnote 77, second reference).

seldom elevated. The blood picture is normal. In three cases in which studies were made, clotting was normal; it was also normal in the case of Burhans and Gerstenberger.[81] The Wassermann reaction is ordinarily negative. The spinal fluid is oftenest slightly yellow, but it may be blood tinged or clear. It is usually under increased pressure. The cells may be increased. Fürstner [69] laid great emphasis on the concurrence of bronchitis. There is occasionally albuminuria. Various forms of mental disease may be imitated, such as traumatic neuroses (Kasemeyer), Korsakow's syndrome [53] or paresis. The patients are often demented, disoriented and irritable. They may be very constipated, and often have retention of urine. Brain tumor or abscess may be closely imitated.

TREATMENT

Spontaneous Recovery.—It is difficult to say how many patients recover. The early reported cases with necropsy, such as those of Textor [16] and of Bouillon-Lagrange,[5] are perhaps open to criticism. In the case mentioned by Bowlby [58] there had been no symptoms. Symptomatic relief, spontaneous or following lumbar puncture, is reported by Curschmann,[82] Dunn,[83] Fried [84] and others, but in none of the cases was the diagnosis verified.

Lumbar puncture is the only definite nonoperative treatment that has been recommended. It was performed in most of the cases subsequently operated on, usually with temporary relief, but in one case (Case 6) with aggravation of symptoms. Fontanelle puncture in infants [43] is of therapeutic as well as diagnostic importance.

Cranial puncture has apparently served to make the diagnosis in some cases and to cure others (Neisser and Pollack,[57] Nonne [52]). But the procedure will scarcely appeal to the modern surgeon in its original form, which has been responsible for several accidents. In one case here reported, the cyst was punctured during the attempt to perform a ventriculogram. The diagnosis was made, but the contents of the sac were too solid to be removed. The procedure might be worthy of trial, however, under certain conditions.

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81. Burhans, C. W., and Gerstenberger, H. J.: Internal Hemorrhagic Pachymeningitis in Infancy, J. A. M. A. **80**:604 (March 3) 1923.

82. Curschmann, H.: Ueber die therapeutische Badeutung der Lumbalpunktion, Therap. d. Gegenw. **13**:241-247, 1911.

83. Dunn, A. D.: Pachymeningitis Haemorrhagica Interna: a Study of Five Cases of Nontraumatic Hemorrhagic Spinal Fluid, Am. J. M. Sc. **163**:819-825 (June) 1922.

84. Fried, H.: Pachymeningitis Haemorrhagica Interna, M. J. & Rec. **119**:149-151, 1924.

Operation.—Meekren [85] in 1519, as quoted by Sprengel,[86] is said to have opened the dura after trephining to allow the escape of pus or blood. This procedure, ignored by Hippocrates, Galen and Fabricius, among others, was condemned by Astley Cooper.[87] Cases in which this measure was resorted to, however, according to Bowen,[51] were recorded by Molleson in 1766 and by Petit in 1790. Hulke [88] seems to be the first modern surgeon to have reported an operation for late subdural hemorrhage. This was in 1881. The patient made a good recovery. Since then, fifty or more cases have been published. Most of them have a marked similarity to one another, and they are well enough represented by the cases herein reported in full. The uncertainty of diagnosis; the color of the tense, nonpulsating dura; the spurt of fluid on incising it; closure with drainage, and the irregular postoperative course repeat themselves from case to case with some monotony.

The problems of the operation differ but little from those of other operations on the brain. Patients have submitted to local anesthesia with surprising self control. Bleeding is seldom abnormally profuse. Whether a bone flap should be raised, or a decompression opening made or the semifluid clot merely evacuated by puncture must depend on the alternative diagnosis and the urgency of the particular case. Recoveries seem to have followed even the simplest procedure (Case 4), but in a case reported by Bernard Sachs and Elsberg [89] little improvement followed partial evacuation of a clot through a decompression, and a bone flap through which most of the clot could be removed had to be elevated twenty days later. On the whole a generous exposure of the lesion by an osteoplastic resection with or without a combined subtemporal decompression to accommodate the subsequent edema would seem to be the preferential method.

Even when the cyst contains clear serum, there may be large clots in it also, and the dura should be widely opened in every case. Guillain [90]

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85. Meekren: Chirurgie, Nuremburg, 1519, Chapter 40, p. 144, quoted by Sprengel (Footnote 86).

86. Sprengel: Histoire de médecine (translated from the German), Paris, 1820, **7**.

87. Cooper, A.: A Series of Lectures on Modern Surgery, Amer. Ed. 1, Boston, 1823, p. 149.

88. Hulke: Severe Blow on the Right Temple Followed by Right Hemiplegia and Coma and then by Spastic Rigidity of the Left Arm; Trephining; Evacuation of Inflammatory Fluid by Incision Through Dura Mater; Quick Disappearance of Cerebral Symptoms; Complete Recovery, Lancet **2**:814-815, 1883.

89. Sachs, Bernard; and Elsberg, C.: Extensive Subdural Hemorrhage after Trauma, New York M. J. **104**:633-635 (Sept. 30) 1916.

90. Guillain: Discussion of case of Phelip: Traumatisme du crâne, Hémorrhagie intra-dure-mèrienne tardive, Trépanation, Guérison, Bull. Soc. de chir. **47**:1033-1039, 1921.

cautions against incising the dura, and Lombard [91] reports a case in which the patient recovered without it, but such cases are much in the minority. In a case in which operation was done by Buzzard,[92] removal of a hematoma from the region of the sinus resulted in a fatal hemorrhage. Usually, however, no bleeding follows the removal of the coagulum from within its organizing capsule, and frequently, as in Case 3, the entire hemorrhagic cyst capsule may be removed in its entirety with but little oozing from the under surface of the dura. Small tags of clot left behind appear to do no harm. Usually, however, the neomembrane over the arachnoid has been left undisturbed, and there seems to be no need of removing it. Such procedures as curettage or excision of the dura, ligation of the meningeal, or the implantation of other tissues appear to be unnecessary, and even harmful, in that they increase the amount of manipulation and prolong the operation. Tamponade of the wound with gauze was practiced by some of the older surgeons, but would scarcely be used nowadays. Irrigation with warm saline solution, sponging with cotton pledgets, and the occasional use of bits of muscle or clips has sufficed to arrest hemorrhage in most cases. The dura may safely be closed, except in severe cases in which a decompression has been made; but if any dural defects are left, the inner layer of neomembrane may serve to protect the brain. Some patients have made a smooth convalescence without drainage, but a small drain does no harm, and oozing may demand its use. The brain usually expands to fill the cavity that is left, and more rapidly if the head of the table is lowered. But in two fatal cases reported by Trotter,[56] the brain failed to regain its size and did not pulsate. A similar condition was seen by Macewen,[93] whose patient recovered.

POSTOPERATIVE COURSE AND COMPLICATIONS

Postoperative Mortality.—It might be supposed that the removal of a subdural hematoma of brief duration would be followed by a complete recovery in all cases. Such is not the fact, however. Three of the ten cases reported here were fatal, as were two of Totter's four cases, and one of the two published by von Saar and Herschmann,[61] all in the absence of gross technical errors. Most of the cases recorded in the literature have been successes but doubtless many less fortunate ones remain unpublished.

Complications.—Patients surviving the operation as long as a week usually do very well. The ordinary postoperative complications, such

91. Lombard, P.: Hématomes intraduraux traumatiques: Inutilité de leur évacuation systematique, Bull. et mém. Soc. de chir. de Paris, **47**:1332-1335, 1921.

92. Buzzard, case cited without reference by Henschen (Footnote 49).

93. Macewen, W.: Cases Illustrative of Cerebral Surgery, Lancet **1**:881-882 1885.

as hemorrhage, infection, pneumonia and nephritis are now seldom seen. The two occurrences that are most to be feared are edema of the compressed brain, and the presence or enlargement of a similar hematoma on the other side. All six of the deaths mentioned above were due to one or the other of these two causes, and they have produced alarming symptoms in many other cases.

Cerebral Edema.—Patients usually seem to be improved immediately after the operation, and make a good ether recovery. That evening, or after an interval perhaps as long as a week, headache, convulsions or coma may set in, or the preoperative symptoms may be reproduced, and even be worse than before. The surgeon's first thought is that bleeding has recurred, but secondary operations and necropsies after various intervals have usually shown that postoperative hemorrhage is comparatively slight, and that the brain is tense, the convolutions flattened, and the ventricle collapsed. This is illustrated in a photograph published by Sachs.[68]

If the first operation has been carefully performed, and drainage has been employed, reexploration should be reserved as a last resort, for it often merely adds to the edema. Lumbar, occipito-atlantoid and ventricular puncture have been tried without success, and seem to possess certain dangers of their own. The use of hypertonic saline solution by vein, by mouth, or by rectum naturally suggests itself, and should certainly be tried; but for some reason its use in this condition has not proved very successful. In cases in which the symptoms are only moderately severe, they may persist for many days.

Bilateral Lesions.—The occurrence of a bilateral hematoma is described by many observers, both in the traumatic and in the spontaneous type. Cases are not rare in the surgical literature in which patients dying after evacuation of a hemorrhagic cyst on one side are found at necropsy to have one on the other also. One such case is reported here. Trotter has published the only two cases in which bilateral operations were performed, one with success. In both cases, an interval of a day or more was allowed to elapse between the two operations, during which time symptoms referable to the unoperated side appeared or grew more pronounced. Trotter believes that this was a mistake, and that both sides should be explored at the same sitting, if there is any suspicion of a bilateral lesion, as evacuation of one side seemed to cause an increase in the lesion on the other. This is probably sound advice. A simple exploratory puncture need not lengthen the operation seriously; but sometimes there is no indication of the presence of the second hematoma.

End-Results.—As a rule, the patients who recover from the operation have done extremely well. There may be slight residual paralyses, headache or vertigo, but complete restitution is the rule. Convalescence

seldom lasts longer than four or five weeks. It is significant that in no case has a return of symptoms been reported. No necropsies on patients dying later from other causes have yet been published, and such cases would be of the greatest interest.

COMMENT ON THEORIES

Traumatic Subdural Hematomas.—There is something very mysterious about the occurrence of these lesions and their delayed relation to trauma. The mechanism of production of the far more common posttraumatic extradural extravasations is well understood, and the process of organization of the clot is the same as that of a hematoma elsewhere in the body. That a clot should slowly form in the mesothelium lined subdural space is quite a different matter. If such clots were likely to follow trauma, one would expect to find them more often after fractures of the skull, and they would be a frequent postoperative complication of intracranial osteoplastic explorations. But as a matter of fact, although a postoperative extradural clot may sometimes form and necessitate the reopening of the wound, the hematoma under these circumstances is almost invariably found between skull and dura—rarely under the dura. It would appear, therefore, that in the disorder under consideration there must be some individual predisposition of the meninges favorable to the subdural seepage of blood.

When such a hemorrhage occurs, moreover, the clot organizes in most peculiar fashion. Naturally in subjects dying a few days after the hematoma has formed, the findings are merely those of a soft, widespread subdural clot. But soon the process of organization begins, chiefly from the dural side. The free surface of the clot furnishes a favorable mechanical support for the migration of the mesothelial cells which rapidly cover it; granulation tissue follows the strands of fibrin, enclosing pockets of serum or cells, and any such spaces, which are particularly apt to occur along the surface of the dura, become lined with a sort of mesothelium. The same sequence of events occurs in experimental hematomas produced in the laboratory.

When the hematoma is very large, its organization may be long delayed, as in an old hematocele, and with fibrosis of its capsule, a serous cyst may be formed. Such a cyst, as in Bowlby's case, may remain permanently symptomless, but it is far more common for signs of cortical irritation to be present.

The appearance of well preserved blood cells and of a layered arrangement of the clot must not be taken as conclusive evidence of successive hemorrhages. This has been pointed out already. Perhaps the majority of the published cases might be explained in this way, and with Trotter one is tempted to force them all without exception into a

single category. But in many cases, the interval of perfect health and full activity is too long to be thus satisfactorily accounted for. The next most simple explanation is that a thrombus gives way, permitting a secondary hemorrhage, or that a trifling oozing becomes more rapid after the patient is convalescent. Such an occurrence, however, has never been observed as a consequence of intracranial operations, when conditions would seem to be most favorable for it, and it could scarcely account for a case such as that contributed by Naffziger (Case 10), where a subdural cyst was observed at a second operation at a spot previously explored and found negative.

In cases in which the symptoms begin at a period when organization must be well established, it is natural to look to the granulation tissue itself for the source of the hemorrhage, as most authors look to the very vascular pachymeningitic membrane in the spontaneous form of the disease. Spontaneous hemorrhage does not occur from even new-formed capillaries in normal subjects, however, nor has it been produced in experimental animals, and we must suppose that there is some peculiarity of the tissue in question, or its environment, which leads to rupture of the capillaries. A similar picture is seen in patients with various hemorrhagic diseases, but in the cases under consideration, no hemorrhagic diathesis has been demonstrated. Postoperative hemorrhage has seldom occurred. The capillaries appear normal microscopically. Rupture of capillaries from obstruction of various kinds has been suggested, but seems physically unlikely. Cultures from the fluid and from the membrane itself have repeatedly been sterile, and organisms are absent from sections. The microscopic picture is not that of an infection. It is significant that the operative wounds stay clean under the conditions of modern surgery, and that meningitis occurred only occasionally in the cases mentioned in early reports.

A feature of traumatic and postoperative subdural membranes which has received little attention is the presence of the lined spaces filled with blood or serum that have been described above. The exact genesis of these spaces is uncertain. They can form anywhere in the hematoma, as is shown by experiments. Their favorite seat, however, is along the surface of the dura, from which they doubtless derive their mesothelial lining. Perhaps they are the remnants of the perivascular channels described by Ford Robertson, and recently confirmed by Jacobi,[94] but their relations are not obvious. Some of them communicate with capillaries in the apparently progressive cases, as has been already mentioned; and if such is the case, it is not difficult to imagine that they might grow like a racemose aneurysm or regress like an occluded vessel, and thus

94. Jacobi, W.: Das Saftspaltsystem der Dura, Arch. f. Psychiat. **70**:268-285. 1924.

give rise to early or late hemorrhages. This theory is founded on microscopic appearances alone and awaits confirmation, as do other hypotheses.

Source of Original Hemorrhage.—The large vessels of the dura lie on its external aspect. The submesothelial layer of capillaries is small and well supported. Significant hemorrhage seems likely to occur, therefore, only after a tear of the dura. The source of immediate subdural hemorrhage has been found in ruptures of the sinus, its lacunae and the tributaries of the veins of the pia covered closely with arachnoid, and of the vessels at the base. Langer [95] reported the presence of veins that leave the brain in the parietal region far from the sinus, but Browning [96] could not confirm their presence. Mittenzweig [97] found such an abnormal vein ruptured in a case of late hematoma of the dura, and discovered fifty-nine similar anomalies in 200 brains that he examined. They occur in lower animals, but in twenty duras examined by us, no communications were found more than 4 cm. from the sinus, as stated by Browning. This need not weaken Mittenzweig's argument, however. The source of hemorrhage is usually not found.

Spontaneous Pachymeningitis.—The origin and nature of the vascular subdural membrane which has been so frequently observed is still in doubt. In a few instances, pus or various organisms have been found in such membranes, but reasons have been given above for believing that their presence is accidental. Nor is the histologic picture that of a true inflammatory process. The name proposed by Virchow is a convenient one, however, and will doubtless be retained. The process has been regarded by Jores and others as neoplastic; but it lacks the property of autonomous growth and differs microscopically from true angiomas. It might be more aptly compared with the telangiectases seen in the skin of old people.

The difference between the structure of pachymeningitis vasculosa and the granulation tissue replacing a hematoma has already been described, and it does not seem possible that the former is due to the simple organization of an exudate, even under the influence of passive congestion. It is conceivable, however, that it is a late, chronic stage of the process, suggested to account for the progression of a traumatic hematoma, namely, the formation of communications between the capillaries of organization tissue and the lined spaces left along the dural

95. Langer, K.: Ueber die Blutgefässe der Knochen des Schädeldaches und der harten Hirnhaut, Denkschr. der Kaiserl Akad. der Wissensch. Mathem. Naturwiss. Classe, Vienna, 1877, quoted by Browning (Footnote 96).

96. Browning, W.: The Veins of the Brain and its Envelopes, Brooklyn, 1884.

97. Mittenzweig: Subdurale Blutungen ans abnorme verlaufenden Gehirnvenen, Neurol. Centralbl., Leipzig **8**:193-196, 1889.

surface, in a manner similar to the canalization of a thrombus. The vessels of the pachymeningitic membrane form a plexus in a manner slightly different from that seen in the reactive subdural membranes, but which may be the result of the establishment of a lively circulation in them.

Perhaps we may trace the formation of a membrane on the internal surface of the dura as follows: The presence of fibrin, alone or in blood or in an inflammatory exudate, causes the proliferation of granulation tissue. The fibrin may be simply organized and regress, forming a fibrous thickening of the submesothelial layer of the dura (pachymeningitis fibrosa). Or microscopic cystic spaces may be included in the membrane, perhaps forming from the perivascular spaces and so surrounding the capillaries, which may form connections with them by rupture or by spontaneous anastomosis. If a circulation becomes established in such spaces, a fine varix is formed (pachymeningitis vasculosa), which is very liable to rupture, giving rise to small or large hemorrhages (pachymeningitis hemorrhagica).

This theory, of course, also lacks confirmation. It fails to explain why the capillaries should form connections with the cystic spaces, and why empty spaces are so seldom observed in the common, nontraumatic pachymeningitis. But other theories are equally incomplete. There is no doubt that many gaps still exist in our knowledge of the nature of subdural hematomas, and it is to be hoped that any facts that may contribute to it will continue to be reported. Of especial interest would be cases in which a true vascular pachymeningitis could be shown to have resulted from trauma; those in which the organization of a sizable hematoma not giving rise to progressive symptoms could be studied, and those in which the late result of operative evacuation of a hematoma could be observed.

REPORT OF CASES

Within a few weeks after the admission to the Peter Bent Brigham Hospital of the patient described in the introductory section of this report, the following two cases came under observation:

CASE 2.—*Subdural hematoma with onset of symptoms five weeks after trauma. Operation three weeks later after lateralization by ventriculography. Osteoplastic craniotomy; removal of clot; stormy convalescence; recovery.*

A Jewish butcher, aged 32, referred by Dr. H. C. Sanders of Claremont, N. H., was admitted, Jan. 27, 1924. He had been born in Russia of healthy parents and had himself previously enjoyed good health. Aside from excessive use of tobacco, his habits were good and he rarely used alcohol.

About Dec. 12, 1923, seven weeks prior to his admission, he was knocked unconscious in an automobile accident, receiving a contusion over the eye and minor injuries elsewhere. He was confined to bed for ten days while recovering from his bruises. He was at this time free from headache.

About Jan. 11, 1924, five weeks after his accident, on returning to his work, he began to be troubled by a left-sided headache, worse on mental or physical

exertion. It was dull and throbbing in character, severe enough to keep him awake at night, and of a character which made him very excitable and restless.

At the time of his admission, the patient was seen to be a well developed and rather spare man, lying quietly in bed. He was cooperative, but easily excited and fatigued. The general physical examination was negative, except for a slight coryza and anosmia.

Examination of the optic fundi showed the disks to be deeply injected, and elevated to a height of about 1 diopter bilaterally. There were no hemorrhages. The pupils were small, and at one examination they failed to react at all. There was a slight clumsiness of both hands in performing various coordination tests. The right knee jerk was slightly more active than the left, and the right hand somewhat the weaker. There were no pathologic reflexes.

The patient ran a slight fever, reaching 38 C. The pulse dropped as low as 46. Routine laboratory tests, including visual fields and stereoscopic roentgen-ray examinations of the skull were negative. One specimen of urine contained albumin.

The headaches were extremely variable, being accompanied at their worst by exacerbations of fever and slowing of the pulse. At these times, the patient was noisy and irritable, tried to get out of bed, refused medication, and, in general, gave his attendants a great deal of trouble.

A lumbar puncture, January 28, yielded 4 c.c. of fluid in rapid drops. It was clear and colorless compared to water. It contained 2 lymphocytes, and showed no increase in protein. The Wassermann and gold solution tests were negative. The patient seemed slightly more comfortable after this puncture.

A tentative diagnosis of hematoma of the dura was made but tumor could not be excluded, and in the absence of definite lateralizing signs recourse was had to ventriculography. This procedure was carried out without incident, February 2, the burr hole was made in the right occipital region. It showed both ventricles displaced about 2 cm. to the right of the midline.

Operation was performed under ether, February 6, by Dr. Horrax. A large, left-sided osteoplastic flap was turned down without difficulty. It was rather more vascular than usual. The dura was under considerable tension and showed a definite greenish yellow tinge. When the dural flap was elevated, a huge organizing clot was seen between the dura and the arachnoid. It covered almost the entire hemisphere and was lined by a membranaceous capsule, which was thicker on the side toward the dura. The capsule was opened, and the semiliquid contents were squeezed and spooned out. As much as possible of the capsule was also pulled away, which necessitated the division of some firm bands between it and the dura toward the midline. They were nonvascular, however. Some portions of the capsule which lay anteriorly and toward the base were not detached. There was considerable oozing from the outer surface of the dura, which was controlled by placement of bits of muscle. The meningeal artery was clipped.

During the painstaking toilet of the wound, the brain which had been much deformed gradually regained its shape. The dura was closed with a gutta-percha tissue drain to the arachnoid, because there was persistent oozing in spite of all measures. The flap was replaced.

The wound drained little, and the wick and stitches were removed in forty-eight hours. In blood taken from the ear, bleeding time was found to be two minutes, clotting time seven minutes. The platelets appeared normal. The patient was fairly comfortable for a few days after the operation, then developed severe headache once more, and was at times irrational. The choking of the

disks persisted, and the bone flap became slightly elevated. Saline purgation had little effect on his symptoms.

His condition, indeed, was most unsatisfactory for a week, when there was a marked turn for the better. He was discharged, February 23, free from symptoms. The choked disks were at this time flat, and there were no positive neurologic findings.

Dr. Sanders reported, Jan. 22, 1925, that the patient had no residual symptoms of his old trouble aside from an occasional mild headache, which does not keep him from work.

Sections from the clot and its coverings taken at the time of operation showed organization of various degrees in progress throughout. The membrane lying on the arachnoid was composed of fresh fibrous tissue, a few cells in thickness, avascular, and covered with a definite mesothelium. Mallory's connective tissue stain showed the entire absence of fibrous tissue in portions of the immediately subjacent clot. The invasion of fibroblasts was extremely irregular, apparently following the framework of fibrin.

The dural surface, on the other hand, showed a comparatively dense and uniform layer of granulation tissue. A striking characteristic of this layer was its extreme vascularity. Capillaries of the usual size were numerous, and in addition there was a series of large lakes of blood enclosed by a definite endothelium, separated by a few layers of cells from the line of detachment from the dural surface (Fig. 5). Tiny, sharp, capillary-like eyes and creeks ran off from these lakes, suggesting a communication between them and the vascular system (Figs. 6 and 7). Other spaces showed fewer red cells and a network of fibrin or granular débris. Some pigment was present. In slides stained with methylene blue and eosin no bacteria were seen.

This case is of especial interest clinically because the patient was a temperate, young man in the best of health. The complete absence of headache up to the time he began work, two weeks after the accident, also is worthy of note. The ambiguous, fluctuant symptoms and the extreme restlessness and irritability, especially after the operation, were characteristic. Attention may be drawn to the diagnostic lateralization of the lesion by the aid of ventriculography; but it may be said that a bilateral exploratory puncture would have served the same purpose, and been more safe.

Pathologically, the resemblance of the large blood filled spaces to the empty lacunae seen in postoperative hematomas (Fig. 8) on the one hand and to the "giant capillaries" seen in nontraumatic pachymeningitis on the other is striking.

CASE 3.—*Subdural hematoma with onset of symptoms ten days after mild trauma and operation eight weeks later. Osteoplastic resection; evacuation of clot; stormy convalescence; reelevation of flap disclosing cerebral edema. Ultimate recovery.*

T. S., aged 58, an Italian café keeper, was referred to the hospital, Feb. 23, 1924, by Dr. Bradford Kent of Dorchester, who related that the man had been in an automobile accident on the preceding December 31, when he sustained a small lacerated scalp wound of the occipital region. He had been rendered unconscious for only a few minutes, and apparently was otherwise unhurt. The scalp wound discharged a little for three or four weeks, then healed.

According to his physician, the patient had always been very emotional and excitable, but not definitely psychopathic. He drank much wine, and was frequently tipsy. Aside from this, the past history was negative.

About January 10, he began to complain of a periodic headache and to walk with a shuffling gait. He was found to have exaggerated deep reflexes, equal on the two sides. The headaches and spasticity increased progressively. Mental clouding was observed early in February, and, February 18, the margins of the left disk were found to be somewhat obscured.

On admission, he complained of a left frontal headache and was very confused and restless. He had no recollection of the injury received three weeks previously, and stated that he had been having headaches for a year. He was unable to give any details of his family or marital history. At times he was

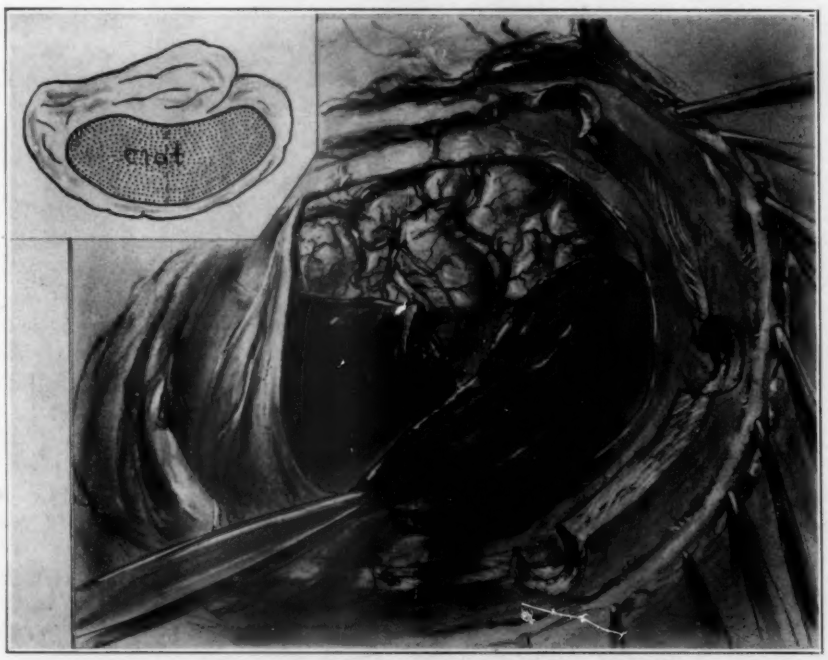

Fig. 12.—Unusually coherent clot being pulled in two large pieces from beneath the dura, from a sketch made at time of operation. Insert shows size of clot in relation to brain.

definitely disoriented for place and would get out of bed and wander about the ward. There was tenderness over the left side of the head. The deep reflexes were variable, perhaps more brisk on the right than on the left. The edges of the left disk were blurred. Visual fields and stereoscopic roentgen-ray examinations of the skull were negative, as were the usual laboratory tests. The urine on one occasion contained albumin.

While under observation, the patient had a slowed pulse, and his temperature tended to be perhaps a little subnormal. The swelling of his disks became gradually more definite, and a height of 1 diopter could be measured March 2. A lumbar puncture was made, March 5, and showed a pale yellow fluid containing 4 cells under increased pressure. The Wassermann reaction was negative in both

the spinal fluid and the blood. The same day, the patient's temperature reached 39.5 C. by rectum, and the white count had risen from 9,800 to 21,000. He became noisy, incontinent and difficult to manage.

A differential diagnosis lay between a left frontal tumor and hematoma, with the possibility of abscess to be considered. An exploratory puncture of the left frontal region was made, March 6, by Dr. Horrax. After a thickened dura was

Fig. 13 (Case 3).—Cerebral surface of the coherent clot, removed complete in two pieces at operation.

penetrated, a small amount of old blood clot was encountered and, at a depth of 3 cm., the needle met definite resistance.

The next day, Dr. Cushing with Dr. Horrax turned down a generous left-sided osteoplastic flap. There was no unusual vascularity. The characteristic olive green of the dura was at once apparent over the upper portion of the exposed field. A dural flap was then raised without breaking the surface of the shiny, greenish, partly organized clot beneath it. The under surface of the dura was

smooth and glistening. The clot after blunt transection to the arachnoid was sufficiently cohesive to be extracted complete in two pieces by gentle traction. It measured 15 cm. in length, 6 cm. in width and about 3 cm. in its greatest thickness. The tissue reminded one irresistibly of the lobes of a mature jaundiced liver, in color, form and consistency (Figs. 12 and 13). No especial bleeding followed its extraction except at its upper posterior margin, where the oozing was controlled by warm saline solution irrigation. The surface of the brain showed a corresponding depression, and the convolutions were flattened and yellowish. The compressed brain filled out to practically a normal contour when the patient's head was depressed. The dura was sutured and the wound closed without a decompression in the usual manner. A single small gutta-percha tissue drain extending down to the arachnoid was left in place for forty-eight hours.

The patient failed to show any definite change in his condition after the operation. His restlessness amounted almost to delirium. He became aphasic and untidy. He was so difficult to control that male nurses had to be procured for him. Liberal saline solution purgation did not help him, and the ordinary sedatives had little effect. The clotting time of blood from his ear was found to be three minutes, bleeding time one and a half minutes; the platelets appeared normal.

Under the apprehension that the clot might have reformed, the bone flap was reelevated, March 10, under local anesthesia. An insignificant, thin layer of clot was found overlying the dural flap. When the dura was opened, a few small tags of clot were found, obviously not sufficient to have caused any symptoms. The brain, however, was rather tight, its surface still yellow, and the convolutions flattened.

This operation also failed to alter the patient's condition and, March 12, he had a generalized convulsion. A few days of mild delirium followed and he then became more quiet. He improved steadily thereafter, in spite of having a large intercurrent carbuncle of the neck which fortunately responded well to treatment by radiation.

On his discharge, March 28, the choking of the disks had entirely subsided; he was talking fairly well and walking without assistance. Four months later, Dr. Kent reported that he was apparently normal in every way. A later report, Jan. 21, 1925, stated that he continued to be well and showed no neurologic symptoms.

At the time of operation, Dr. Cushing removed a block of tissue for study. It included the dura and the entire thickness of the clot and its enveloping membrane intact in one piece. Sections showed the dura to be of normal thickness and appearance. There was no widening or atheroma of the vessels, and no infiltration of the tissue. There was a definite layer of large, elongated spaces, separated from the dural surface by a narrow, uniform band of new fibrous tissue (Fig. 4). These spaces were lined with a mesothelium which differed from the endothelium of the capillaries in minor respects; the cells were more irregularly placed, more sparse and flatter. There was no trace of adventitia even about the largest spaces. They contained some red cells, but were not as full as most of the capillaries. There was a definite fibrin network in some portions of them. Small buds and bays ran out of them, suggesting a communication with the capillaries.

The remainder of the dural membrane was thin and contained a few capillaries of normal size. It stopped rather abruptly at the ege of the clot, which was only slightly invaded by fibroblasts, as shown by Mallory's connective tissue stain. The center of the clot was mainly composed of decolorized red cells, but fresh highly stained ones were scattered through it. In some areas, fibrin was almost entirely absent, and no wandering cells were seen. The clot, and to a less degree its limit-

ing membranes, was diffusely infiltrated with leukocytes, to a greater extent than in any of the other cases of this series, or than those illustrated elsewhere. No bacteria were seen in sections stained with methylene blue and eosin. Only shreds of fibrous tissue were seen on the arachnoid surface, the greater part of the inner neomembrane having apparently been detached and lost during fixation and sectioning of the block of tissue studied. A moderate amount of pigment is present.

This patient was moderately alcoholic, and had shown some signs of a psychopathic personality before the accident. The clinical course was characteristic, as were the findings on puncture. In few cases has the clot in similar fashion been removed in its entirety and almost intact. The postoperative course is of interest, in showing that because of cerebral edema the preoperative symptoms may become actually accentuated. It also shows that reexploration need not necessarily be undertaken in view of this fact. The existence of a contralateral hematoma though not excluded was improbable. The presence of an unusual number of leukocytes in the hematoma may perhaps be accounted for by the associated leukocytosis of the carbuncle which was already forming.

In addition to the foregoing three typical examples of subdural hematoma, two other cases have been found in the early records of the Peter Bent Brigham Hospital and it is possible that there may be still more hidden under obscure diagnoses and in cases in which the lesion was not verified by operation or necropsy. The following case, for example, operated on by my predecessor, Dr. Kenneth McKenzie, now of Toronto, had been diagnosed as a "cerebral tumor suspect: arachnoid cyst."

CASE 4.—*Subdural hematoma disclosed in the process of ventriculography for an obscurely localized intracranial lesion, supposedly a ventricular tumor. Partial evacuation. Recovery. History of trauma subsequently secured.*

S. S., a Hebrew laborer, aged 38, was admitted to the medical service, Aug. 31, 1923, the diagnosis being cephalalgia and constipation. He had had periodical headaches all his life; indeed, they were said to be familial. They were ordinarily relieved by powders, but a week before his admission they became more severe than usual and caused nausea and vomiting.

The physical examination was essentially negative except for very sluggish knee jerks, a questionable nystagmus and weak convergence of the left eye. Owing to lack of cooperation, a lumbar puncture was performed with difficulty. The fluid thus obtained was slightly bloody. The Wassermann reaction was negative. He was discharged.

September 19, he was readmitted to the medical service because of an increase in his symptoms. His headaches were most severe, requiring narcotics. There was evident diplopia; suggestive nystagmus, and absent abdominal reflexes. Knee jerks were not obtained. The pupillary reactions were sluggish. Many possible diagnoses were considered: encephalitis lethargica, intestinal intoxication, tubercular meningitis, cerebral tumor and hemorrhagic pachymeningitis, in view of the bloody tap on his previous admission. A second lumbar puncture showed a

xanthochromic fluid with 14 mononuclears, 2 polymorphonuclears and globulin. He was transferred to the surgical service, September 27.

He was drowsy, dazed, moaning and uncooperative. He complained of unbearable suboccipital headaches, and there was suboccipital tenderness. The optic disks were normal; the right pupil was slightly larger than the left, and knee jerks were absent with reinforcement. All other reflexes were present but were sluggish and difficult to elicit. Roentgen-ray examination was negative.

October 1, Dr. McKenzie set out to inject air into the right ventricle for purposes of ventriculography. Under local anesthesia, an opening was made with a burr through the skull in the right parietal region. The exposed dura was bluish, and for a time it was supposed that there was a large underlying vein. The membrane was finally opened and a mixture of yellowish fluid and old blood clot escaped. When this cavity was gone through, a whitish, tough membrane was met on the cerebral surface. The needle was carried through the membrane to the ventricle and 10 c.c. of slightly xanthochromic fluid was withdrawn and supposedly replaced with air. The subsequent films showed the ventricles to be apparently normal in outline but definitely displaced toward the right, the side of the lesion. Much of the air had escaped into what was evidently a large subdural cyst beneath the cranium.

This procedure had two interesting sequels: one, it completely relieved his headaches; the other, it led to questioning which brought out the fact that he had been hit with a baseball bat on the right parietal occipital region some time in June so far as he could recall, viz., about four months previously.

He refused any further treatment; indeed, none seemed necessary. He was discharged, October 9, free from symptoms and, according to reports, remains well two years later, aside from the periodic headaches he has had all his life.

There can hardly be any question as to the diagnosis in this case. It is remarkable in the prompt relief of symptoms afforded by the very incomplete evacuation of the semifluid contents of the hematoma. If such a trifling procedure should suffice to cure such a condition, the major operations described in the preceding cases would appear to be unnecessary. However, it would seem preferable to remove the organizing envelops of the clot when this is possible. It will be interesting to follow a case such as this to the end and to learn what is the ultimate fate of the lesion. In all likelihood, it would be indistinguishable from the laminated pachymeningitis interna of Virchow.

One other matter is of considerable interest in this case: namely, that the ventriculogram which, under the circumstances, hardly needed to be taken nevertheless showed the ventricles to be dislocated *toward* rather than away from the side of the lesion.

The following case, the earliest identified in the hospital files, was the occasion of an operative fatality. One is led to consider, in view of the foregoing history of Case 4, whether a less radical procedure, such as the mere tapping of the cyst and the withdrawal of its contents by suction, would not have been a more correct temporary procedure at least. This, however, was not possible as no exact pathologic diagnosis had been made.

CASE 5.—*A nontraumatic (?) subdural hematoma of seven weeks' duration with marked psychosis. Supposed tumor. Operation; fatality; cerebral edema and bilateral hematoma.*

N. A., an American business man, aged 57, referred by Dr. C. D. Brooks of Detroit as a cerebellar tumor suspect was admitted, April 4, 1917. He had always been healthy. He was a moderate drinker. No history of accident was obtained.

For some six weeks prior to his admission, after some languor and restlessness, he began to complain of frontal headaches coming on in the evening. Two weeks later, they shifted to the occiput and left side, becoming more and more severe and increased by any movement to such an extent that he would groan aloud. For a week he had had some ataxia, tending to fall to the right and backward. He also had frequent prolonged attacks of sneezing.

Ten days before his admission, definite mental deterioration was noticed. He kept getting out of bed "to find money to pay the barber with." He had at times been so noisy and restless that forcible restraint had been necessary. For four days he had twitching of the right arm. A lumbar puncture showed a clear, colorless fluid under high pressure containing 78 large lymphocytes and an excess of globulin. The Wassermann reaction was negative in the fluid and also in the blood.

On his admission, he was stuporous, cyanotic and unable to speak. Both fundi showed edema and slight blurring, but cupping was present; the laminae were obscured and the veins were tortuous. The blood pressure was 123 systolic, 90 diastolic. A roentgen-ray examination of the skull showed enlarged venous channels. The following day he was jovial and wide awake, but completely disoriented, irrelevant, untidy and obscene. A male nurse had to be provided. He had a definite aphasia; he was able to name objects only by their qualities (such as calling an electric bulb "glass"), and was unable to write more than meaningless syllables. Aside from these mental symptoms, a neurologic examination showed little beyond a marked static instability, and on occasion the left pupil was observed to be smaller than the right. Routine laboratory tests were negative.

The patient was left-handed, and under the presumption that he had a right frontal tumor Dr. Cushing operated, April 16. There was excessive bleeding during the elevation of the osteoplastic flap. The dura was discolored. When it was incised, a large amount of old semifluid blood escaped. This was contained in a great cyst, the outer surface of which adhered to the dura from which it could be stripped away. Over the greatly depressed brain was another dura-like membrane, which covered without attachment almost the entire surface of the hemisphere. Under the belief that the oozing surface of the vascularized dura would serve merely to reproduce the clot, the exposed part of the membrane was cut away and bleeding from its edges prevented by the placement of a series of silver clips. The lower neomembrane was left to serve the purpose of the dura. The bone flap was replaced without decompression, and the wound closed without drainage.

The patient seemed in good condition at the end of the operation but never recovered consciousness sufficiently to speak. He responded to stimulation by opening his eyes and moving his extremities equally and freely. The pupils were equal. The following morning he was much worse. There were no localizing signs. The temperature rose rapidly to 41 C., and the patient died.

Necropsy was performed two hours postmortem. It was limited to the head. The brain was removed in the usual fashion. There was no secondary hemorrhage at the site of operation. The brain, however, was edematous and the convolutions greatly flattened. Except for the oval of dura about 4 by 7 cm. which had been removed, the envelop of the hematoma was complete.

Over the left hemisphere, there was found an intact hematoma containing old fluid and otherwise exactly similar and of equal size to that disclosed at operation. Careful examination revealed several small fibrous bands running from arachnoid to neomembrane, but none of them were vascular and no ruptured vessels were found. The edge of the hematoma reached the pacchionian bodies and the lateral expansions of the sinus, but no lesion could be demonstrated here. The vessels were not particularly atheromatous. There were no other lesions.

Sections taken from the edge of the intact hematoma on the left showed a thin dura, with a narrow lining of granulation tissue, not very vascular. No mesothelial lines spaces were seen. A narrow zone of clot contained collagen fibrils, but no blood vessels. It was covered by a very delicate neomembrane, in places scarcely distinguishable from the arachnoid. No bacteria were seen in sections stained by Weigert's method. Small, thin tags torn from the layer next the dura when stained and cleared showed only cells, fibrin and occasional fibroblasts.

This patient was not the sort in whom one would expect to find a spontaneous pachymeningitis nor did the pathologic appearances resemble it. A trauma may have passed unnoticed. The clinical course and operative findings were typical of subdural hematoma. Later experience has shown that it was unnecessary and perhaps unwise to excise a portion of the dura, as was done here. The surgeon's apprehension of subsequent bleeding from the inner surface of the dura when it was denuded of adherent clot seems to be largely unfounded. The aggravation of symptoms following operation might have suggested a contralateral process, as in Trotter's case, but other patients showing as severe reaction have eventually recovered without further operation. However, owing to the not infrequent bilaterality of the process, it might not be amiss for surgeons to make it a rule always to make an exploratory puncture over both hemispheres when a subdural hematoma is suspected or when a unilateral lesion has been disclosed.

The gross necropsy findings were not in any way unusual. The microscopic appearances possibly suggest a recent hemorrhage, perhaps outside the bounds of the original pathologic process. The sections showed very slight vascularity of the neomembranes, and the absence of mesothelial lined spaces was unusual.

CASE 6 (Contributed by Dr. Ernest Sachs of St. Louis).—*Subdural hematoma simulating frontal tumor, disclosed in the course of a subtemporal decompression five months after a cranial trauma.*

A negro farmer, aged 58, was transferred from the medical service, Nov. 29, 1919, with the following history: In the preceding July, he was thrown from a car and remained unconscious part of the day. What followed is not recorded, but four months later, that is early in November, he was sent to a local hospital because of his mental condition. He was forgetful, irrational, and complained of dizziness. While there, a lumbar puncture was made, following which the symptoms became very much worse, and he had irregular breathing and increased stupor. The findings in the fluid are not known.

After his removal to the Barnes Hospital, November 27, he was found to be apathetic. He answered questions with difficulty, but seemed to realize his sur-

roundings. The pupils were equal, regular and reacted to light. He either could not or would not raise his left arm. When the right arm was raised, there were convulsive movements of the fingers and hand. There was stiffness of the neck. The arms were spastic but no pathologic reflexes were demonstrated in arms or legs with the exception of a Babinski reflex on the left side. Eyeground examination showed a choked disk with retinal hemorrhages in the right eye; in the left eye, beginning atrophy. The systolic blood pressure varied between 100 and 60. With the concurrence of Dr. Schwab, the patient was transferred for operation.

A diagnosis of a brain tumor in the last stages of compression, probably of the right frontal lobe, was made. As the Babinski reflex was on the left side, a right subtemporal decompression was made by Dr. Sachs. The dura pulsated freely but had a bluish appearance. When it was opened, huge quantities of fluid that looked like an old blood clot escaped. The cortex could not be seen and on further exploration it became apparent that a hemorrhagic membrane lay between the dura and the pia arachnoid. As much as possible of this membrane was removed and the usual decompression was done.

The patient did not rally from the operation. Coma increased, and believing that there was a secondary hemorrhage, Dr. Sachs reopened the wound the same night. A further quantity of old blood mixed with cerebrospinal fluid escaped. There seemed to be no active bleeding. The wound was closed. A small rubber tissue drain was left in the wound. The patient died the next day.

Necropsy showed only a trifling extravasation at the site of operation. The envelop of fibrous tissue that had enclosed the clot was still in position. The most striking feature of the gross examination was the extraordinary edema of the right side of the brain, which had elevated the cortex to its original position, and had almost obliterated the ventricle on that side. (A photograph of this brain has been published elsewhere.[68])

A specimen of the dura and hemorrhagic membrane removed at operation, which Dr. Sachs kindly forwarded for comparison with the others, showed a clot composed of red cells in a fibrin network of varying coarseness, becoming organized from the dura. Fresh sprouts of connective tissue traversed the thickness of the clot. Capillaries were moderately large and numerous. There was a definite demarcation between the dura and the granulation tissue zone. About at the junction of the two lay a series of elongated, irregular spaces, lined with mesothelium and containing many red blood cells and a light fibrin network. Few leukocytes were seen. The dura appeared normal.

An interesting clinical feature of this story is the aggravation of symptoms following lumbar puncture which, to judge from other cases in this series, usually gives some relief and has in some reports been stated to be curative. Also a choked disk of such an advanced degree is most unusual. One would have supposed that the operative procedure that was carried out, provided a sufficiently large decompression was made, would have been the most favorable one to combat the subsequent edema of the brain following the release of pressure—a condition that appears to have caused death.

CASE 7 (Contributed by Dr. A. W. Adson of Rochester, Minn.).—*Subdural hematoma simulating tumor three months after trauma. Ventriculography. Operation with excision of bone and dura. Recovery.*

A Norwegian farmer, aged 46, came to the Mayo Clinic, June 15, 1923, complaining of right-sided weakness, headaches, temporary blurring of vision and

vomiting for about two and one-half months. The family, marital and past histories were negative as obtained from the patient; but later the patient's neighbor stated that he had been queer for five or six years and had been committed to the local state hospital twice, four and one-half and three and one-half years previously for two and three months, respectively. He had acted peculiarly, suspected neighbors of doing him injury and his wife of infidelity; he was often melancholy with a tendency to religiosity. He had no diseases of importance. The patient denied any injuries, but after the operation the history was obtained from his wife that three months before admission he had been kicked by a colt, the hoof striking on the left side of the head. He was knocked to the ground and was semiconscious for a few minutes and was carried to the house, where he remained in bed for two or three days. After that time he went about his regular work and had completely forgotten the incident.

The patient's present illness began two and one-half months previous to his admission, two weeks after the injury, with weakness in the right leg and knee which very nearly resulted in his falling on many occasions. About two weeks after this, frontal headaches developed which would awaken him about 4 a. m., and last about an hour. He vomited two or three times a week. There was little nausea, but apparently the vomiting was not projectile in type. He had some pain in his eyeballs and blurring of vision which became progressively worse, so that just before coming to the clinic he was unable to drive his car. There was no history of ptosis, diplopia or of hallucinations.

While obtaining the history from the patient some indications of mental change were apparent. He would interpose irrelevant remarks about his farm, about the Bible, the "spirit," salvation, damnation and so forth. General examination revealed a well nourished adult male with good color, pupils normal; the teeth were carious. Examination of the head, neck, chest, abdomen, genitals, rectum and extremities were negative. The systolic blood pressure was 118 and diastolic 70; the pulse was 72. The spinal fluid showed 2 lymphocytes; the Wassermann, gold curve and other tests were negative. Roentgen-ray examinations of the chest and lumbar spine were negative. The eye report revealed a vision of 6/15 in the right eye and 6/10 in the left; the pupils, reflexes and fields were normal; examination of the fundi showed edema of the disks of about 3 diopters with hemorrhages and exudates and simulation of macular stars. There was a small corneal scar on the right. The Bárány test was negative.

The neurologic examination revealed slight horizontal and vertical nystagmus on the right. Hearing was very poor, especially on the right side. There were no abnormal motor or sensory signs. The reflexes were somewhat irregular with slight increase of the biceps jerk on the right, of the Achilles reflex bilaterally but more on the right, and slight diminution of the patellar reflex bilaterally. There was very slight loss of coordination in the fingers and toes on both sides. The bladder sphincter was somewhat relaxed. The patient was definitely below normal for intelligence, memory, attention and cooperation. No nasal pathology was found.

A diagnosis of a lesion in the left side of the brain, probably in or near the frontal lobe, was made, but as the location was not definite a ventriculography was advised and, July 9, this was done in the usual manner under local anesthesia. When the dura was opened on the left side, a dark vascular mass was encountered; the opening was enlarged forward for about 1 inch (2.5 cm.). When a trocar was inserted into the mass, it was found to be quite resistant and felt like a firm glioma. It was thought advisable to complete the ventriculogram and to do an exploration later. This was done and the films showed a mass in the left parietal region pushing the ventricles downward.

An operation was advised and was performed, July 16, under local anesthesia. Instead of turning a bone flap, skin flaps were turned and the bone rongeured away around the trephine opening, leaving a defect about 3 inches (7.6 cm.) in diameter. The dura was opened, revealing a large, reddish mass which proved to be a hematoma. About 60 c.c. of old clotted blood was removed and a portion of the dura was excised. The bone defect was left uncovered to be repaired later if necessary. A trocar inserted in various directions into the brain revealed no masses or tumors. Colorless cerebrospinal fluid escaped through the cannula inserted into the posterior horn of the ventricle.

The patient's convalescence was uneventful, and the wound healed by primary intention. The disks gradually subsided. About two months after operation, examination of the eyes revealed a vision of 6/7 in the right eye and 6/6 in the left. The fundi showed marked hyperemia of both disks, the right more elevated than the left; the blind spots were enlarged but less than formerly. A letter received from the patient eight months after the operation stated that he was able to do his work normally, that his vision was apparently normal, but that he had not completely cleared up mentally; he still worried a great deal.

This is a perfectly typical example of subdural hematoma. Excision of the dura (also Case 5) is probably not essential, but, as this patient's unusually smooth postoperative course shows, is not detrimental. It is possible, indeed, that the removal of both bone and dura in this case with closure of the scalp alone over the defect may have acted as a decompression and served to forestall the postoperative symptoms which, attributed to edema, have occurred in other cases in the series.

CASE 8 (Contributed by Dr. A. W. Adson).—*Subdural hematoma with onset of symptoms three months after a mild trauma and operation four months later. Removal of clot with decompression. Recovery.*

An Italian cook, aged 30, came to the clinic, July 24, 1923, complaining of headaches, blurring of vision and vomiting for about four months. The family history was negative. He had had gonorrhea in 1914, and influenza in 1917. He denied having had any injuries until after the operation, when he remembered that he had fallen on the ice seven months before admission. He was unconscious for ten minutes or so when a passerby found him, aroused him and assisted him to a nearby house. He recovered completely the same day.

Except for this one accident, the patient was perfectly well until five months prior to admission when he had pain in the right ear for a day followed by a bloody purulent discharge which lasted for about a day. Immediately following this, the patient had a tonsillectomy performed followed by some deafness which disappeared in about a week. Four months prior to his admission, he developed severe occipital headaches with blurring of vision and vomiting; the vomiting occurred three or four times daily for two or three days and was projectile in character.

The headaches came on daily for almost a month beginning about 7 p. m. They lasted all night and until about eleven the next day; the patient was so weak that he remained in bed. Later he was taken to a hospital where he remained one month. His symptoms persisted and he finally left the hospital without improvement. About this time, two months before coming to the clinic, the headaches disappeared and he had had none since, but the blurred vision continued and he was troubled with dizziness. He says that he reeled at times like a drunken man. There was diplopia for two or three weeks. These symptoms then disappeared

but the headache continued, and he complained of being quite nervous and irritable. He was given two injections of neoarsphenamin intravenously without improvement. There was no history of convulsions, motor or sensory disturbances; he never had difficulty in speech or swallowing, and no incontinence of bladder or bowels.

On his admission, the general physical examination was negative. The systolic blood pressure was 116, diastolic 64, and the pulse was 72. Routine laboratory examinations, including a Wassermann test of the blood, were negative. The nose showed septal deflection on the left with some mucopus; the ears were normal and the nasopharynx showed some pus on the right side. Roentgen-ray examination of the head was negative. The eye report showed a vision of 6/15 in each eye; the pupils, reflexes and fields were normal; the fundi showed bilateral choked disks of 2 diopters on the right and from 1 to 2 on the left; there were some hemorrhages and exudates around the disks. The blind spots were enlarged. The Bárány tests were negative. The neurologic examination revealed slight horizontal and vertical nystagmus of both eyes; the examination was otherwise negative.

A diagnosis was made of a probable posterior fossa lesion not very well localized. A ventriculography was advised and, August 7, was performed in the usual manner. The films showed a mass in the left parietal region.

August 13, under local anesthesia, a left osteoplastic flap was turned and when the dura was exposed it was found to be dark and very vascular. When this membrane was opened, it appeared that the arachnoid was thickened to about 1 mm., and under this was a degenerative mass that proved to be an old blood clot. This extended over the left temporal and motor area and forward over the frontal lobes. The cortex had been pushed about an inch (2.5 cm.) from the dura by the mass. The old blood clot was removed and the dura left open and covered with animal membrane. A large decompression was made.

The patient made an uneventful convalescence, and the wound healed by primary intention; the eyesight improved steadily. Three weeks after the operation, the vision was 6/6-3 in each eye; the pupils were equal and the reflexes were normal. The fields were negative and the fundi showed receding choked disks of 1 and 2 diopters; the patient read normally. An attempt to obtain a report of the patient's condition in June, 1924, was unsuccessful.

This also appears to have been a typical case, though the intercurrent otitis media must have been confusing. Apparently in both the foregoing cases the relation to trauma was not elicited until after the operative findings prompted questions in this regard. The choked disk in each of them was higher than appears to be usual with these hematomas, and it is natural that tumor should have been suspected.

Convalescence in both of Dr. Adson's cases would appear to have been more uneventful and rapid than in most of the other cases herein reported, and this may have been due in part to the removal of bone and dura. In other cases in the series, as may be observed, the osteoplastic flap was made and replaced, and, though the ultimate result was perfect, a more stormy convalescence was recorded. In one of them (Case 3) the flap was reelevated; and in the following case also a secondary operation was necessary.

CASE 9 (Contributed by Dr. H. C. Naffziger, San Francisco).—*Subdural hematoma with symptoms of onset eight weeks and operation thirteen weeks after*

trauma. Secondary operation with removal of dura and bone with fascial graft. Recovery.

An American motorman, aged 50, was admitted to the University of California Hospital, June 9, 1914. He had always used alcohol and tobacco to excess. He was disabled by rheumatism at about the age of 25 and his heart was affected at that time; at 45, he had malaria. He had led a hard, irregular out-of-door life, and stated that he had both gonorrhea and syphilis.

Early in April, 1914, he was held up by robbers and knocked unconscious by a blow on the right side of the head. On recovering an hour later, he was able to get up and walk home. Since that time he had not felt like working and had noticed that the left side of his face was weak. About May 29, he began to have a severe, continuous ache in the right side of the head, chiefly in the occipital region. His eyes ached at first, so much that he could not read. June 5, he noticed a weakness of the left arm and leg, which steadily progressed up to the time of his admission. He slept poorly. He believed his memory was unaffected.

On admission he was seen to be a rather fat, well developed man, drowsy and stupid, but able to answer questions. The left side of the face drooped slightly. The pupils were small, the left larger than the right. There was spasticity and weakness with slight hypesthesia of the left side of the body. The heart was enlarged and its action irregular. The Wassermann reaction in blood and spinal fluid was negative. The fluid showed 7 cells. Other tests were negative, as were the routine laboratory examinations. Fields could not be obtained on account of the patient's dulness. There was slight blurring of the optic disk.

While he was in the hospital, his condition varied but tended on the whole to become worse. At times he was somewhat stuporous and had short waves of periodic respiration. His pulse sometimes fell below 50. The spasticity increased. Occasionally he would rouse from his stupor and be able to talk normally.

June 22, an operation (Dr. Naffziger) was performed under gas and ether. A small flap was turned down in the region of the right motor cortex. The dura was found to be tense and bluish. When it was incised, it proved to be about 3 mm. thick. Brownish fluid at once spurted out. On further investigation, a large cyst about 10 by 12 by 5 cm. was found, lying between the dura and the arachnoid. It extended from the frontal to the occipital region, and from the longitudinal sinus to below the temporal ridge, and had produced great excavation of the cortex. It was surrounded by a thin membrane and contained black clots in addition to brownish fluid. The partially organized layer of clot on the inner surface of the dura, about 1 cm. in thickness, was partially scraped away. The middle meningeal artery was tied. A decompression opening was left and the wound drained.

The patient made a rapid and satisfactory ether recovery and was able to move the left side well. There was a moderate amount of drainage for two days. It then ceased with a return of some stiffness of the left side.

On the fifth day postoperative, he had a typical left-sided jacksonian attack. He was ordered at once to the operating room. When the flap was again raised, it was found that there was a clot, about 3 mm. thick, between the dura and the bone. The dura itself had increased in thickness to more than 1 cm. The bony defect was enlarged with rongeurs, and as much dura as possible was excised from the motor region. A fascial flap from the thigh was sutured in its place. The cranial portion of the flap was removed and the scalp closed with drainage.

Following this operation the wound drained profusely for a week, then less profusely for ten days. The patient improved progressively and had no more seizures. He was discharged, July 25, walking and using his left arm and leg apparently without disability.

When seen, March 15, 1921, seven years later, he appeared to be in excellent condition. There was no weakness of any of the extremities. His memory was poor. There was some pallor of the disks and tortuosity of the vessels, but no swelling.

Cultures on agar from the cyst fluid were negative. There was no microscopic report on the excised dura.

The history and operative findings are sufficiently typical. Of interest is the apparent increase in the thickness of the dura between the two operations. It is difficult to believe that this could in itself have been a source of the recurrence of symptoms. As in Cases 5, 7 and 8 of the series, the dura was removed as the presumable source of symptoms. It may be noted, however, that at the reexploration no fresh underlying clot was found, and the experience with other cases in the series would make it appear that dural excision is an unnecessary precaution.

CASE 10 (Contributed by Dr. H. C. Naffziger, San Francisco).—*Subdural hematoma with onset of symptoms a year after a severe trauma. Successive operations with disclosure of the lesion and ultimate fatality.*

J. B., an Italian fisherman, aged 34, a moderate user of wine and whiskey, was admitted to the University of California Hospital, May 20, 1919. Sixteen months before, i. e., Jan. 1, 1918, he had fallen from a pier, landing on his head on a rock. He remained unconscious for twenty-four hours, and was taken to the Lane Hospital, Monterey, where, after removal of some fragments of bone, his lacerated scalp was sutured. He was in the hospital for seventeen days and was discharged in good condition. He went to work at once and continued without symptoms for a year. He then began to have attacks of dull pain in the frontal, occipital and left parietal regions accompanied by dizziness, lasting ten or twelve days at a time. His vision became blurred, especially during the attacks of headache. The symptoms had become distinctly worse the week before his admission. He did not vomit and had no convulsions. He had loss in the sense of taste.

On admission he was seen to be a well developed and nourished, apparently intelligent man, speaking broken English. His pupils were regular and reacted normally, but the left was larger than the right at times. There was a bilateral choked disk, with tortuosity of the vessels. The blood pressure was 110 systolic, 70 diastolic. The deep reflexes were active but equal on both sides. The spinal fluid and blood Wassermann and routine laboratory tests, including perimetric fields, were negative. The temperature was occasionally slightly subnormal. A spinal puncture had been done elsewhere and the fluid was reported normal. Roentgen-ray examination showed a bony defect in the frontal region with an atrophic area just posterior to it. It was supposed he had a subacute abscess, or possibly a tumor.

He was operated on, May 25, by Dr. Naffziger under gas and ether. The old wound on the right side of the forehead was first explored. Beneath it, a bony defect about 3 cm. in diameter was found, bridged over by scar tissue. This was incised and the dura opened. The brain appeared normal, but the meninges were edematous and exuded fluid. No pus was found on needling. The ventricle was entered, however, and 10 c.c. of clear fluid was withdrawn. The wound was closed without drainage.

The head was then turned on the left side and a typical subtemporal decompression was performed, an opening 1.5 by 6 cm. being made. The dura was opened widely. Just above the external auditory meatus a bluish-looking portion of brain tissue appeared, which was considered to be a tumor. When this area was punctured, only blood was obtained. The wound was closed without drainage, the dura being left open.

He made a good ether recovery but was very restless and difficult to control. He had to be catheterized. The later postoperative course was satisfactory. He was discharged, June 10, feeling well. The disks were lower but with margins still obscured.

Eighteen months later, Dec. 2, 1919, he was readmitted to the hospital. At that time mental impairment was definite, although the patient denied having headache, visual disturbance or other new symptoms. Examination was unsatisfactory on account of lack of cooperation, but the disks were more swollen than at the previous admission and the deep reflexes much more lively. There was bilateral ankle clonus. The hernia through his decompression seemed more tense.

While under observation he was often semistuporous and had difficulty in speaking, according to an interpreter. He was noisy and incontinent. He ran a slight fever, reaching 37.8 C. rectally. The white count was 10,200. He was given a roentgen-ray treatment without definite result. The left side of the body seemed more spastic than the right, and a positive Babinski reflex and Oppenheim sign appeared.

As his condition continued to grow worse, a second operation was undertaken, December 23, under gas and ether. The old right frontal flap was raised. The dura was found very dense. When it was incised there was a jet of straw-colored fluid, a large amount of which escaped. The dura was then turned back and was found attached to an underlying membrane by columns of tissue about 1.5 mm. in diameter. These columnar attachments were composed of a tissue denser than pia arachnoid, and there were perhaps ten of them in the area uncovered. "As one looked into this space (Dr. Naffziger writes), it was as if looking into a ruin of some kind, with many white pillars." They were cut without bleeding and it was then seen that there was an additional thin, organized membrane overlying the pia arachnoid. This was incised and resected. The brain beneath showed some flattening of the convolutions but was otherwise normal. There was no free hemorrhage. The ventricle was punctured and much fluid withdrawn. It contained only 2 cells per cubic millimeter and was otherwise negative. The dura and the remainder of the wound was closed without drainage.

The patient left the table in good condition. That afternoon the pulse and respiration rose rapidly and he developed Cheyne-Stokes respiration. The flap was found to be under great tension. Later the same day, therefore, the wound was reopened. It was found to be in very satisfactory condition. There was no bleeding of any consequence, but the brain was again under extremely high pressure, and on ventricular puncture a large amount of clear fluid spurted out. As much was allowed to escape as possible, a drain was introduced beneath the dura, and the wound was again closed. The patient seemed somewhat improved. A spinal and an occipito-atlantoid puncture were performed but secured little fluid. On his return to the ward his respirations were lower and more regular, but the pulse became progressively weaker. In spite of stimulation, he died two hours after the third operation. Permission for necropsy was refused.

Dr. G. Y. Rusk reported that microscopic examination of the tissue showed a dense layer of connective tissue, in the meshes of which were several large vessels. On the inner surface of the layer of connective tissue was an extensive hemor-

rhage. The hemorrhage in part had a laminated structure and pale areas, apparently platelets, associated with accumulations of polymorphonuclear leukocytes ran in irregular lines through the groups of red cells. In other sections the hemorrhage was not nearly so extensive, but here it was intersected by a fine overgrowth of fibroblastic elements and endothelial cells, the latter forming fine capillaries. The diagnosis was hemorrhagic pachymeningitis with beginning organization.

The remarkable feature of this case lies in the fact that a subdural cyst from an old hematoma was finally disclosed at a spot that had been explored and found to be essentially normal about six months previously, and almost two years after the accident that was the probable cause of it. The old scar at the site of the original compound fracture must have given surgical difficulties; but since Dr. Naffziger felt convinced that the cyst could not have been overlooked at the first operation, it would suggest that the process was very late in forming and that the symptoms leading to the first operation would have to be ascribed to some other cause.

It is possible, of course, that this moderately alcoholic patient already suffered from a pachymeningitis, that the fall had nothing to do with his symptoms, or merely precipitated them, and that the mass in the temporal region was a tumor after all. But this seems improbable. The story in retrospect is fairly characteristic of subdural hematoma except for the fact that the initial trauma was more severe than that described in any other other of the reports. The postoperative upset with incontinence and delirium is also typical. As in other cases, no postoperative extravasation of blood was found to have taken place at the secondary exploration, the symptoms apparently having been due to edema.

CASE 11 (Contributed by Dr. H. C. Naffziger).—*An obscure case of organized subdural membrane in a child with cerebrospinal rhinorrhea and hydrocephalus following trauma. Operation: recovery.*

The patient was a boy, aged 20, hydrocephalic from early life and not too bright mentally. He was undersized and showed some evidence of polyglandular dystrophy. He was, however, considered normal by his family.

Two years before his admission, a moderately severe head injury was followed by a rhinorrhea that persisted until four months before admission. On its cessation, he became distinctly more dull mentally. He showed no other new symptoms for several weeks, when he developed a bilateral paralysis of conjugate movement upward.

On admission to the University of California Hospital, it was found that his optic fundi showed an old atrophy but no swelling. Ventricular puncture restored the full range of motion to his eyes for about twenty-four hours.

It was supposed that the patient had had an internal hydrocephalus since early life, but had for a long time overcome his obstruction. After the establishment of his leak and then its stoppage, he had again begun to show signs of pressure. A pineal tumor also was considered a possibility. Dr. Naffziger made an occipital flap, planning to make a ventricular communication with the cisterna ambiens in the event of finding no growth.

A thin layer of clot was found subjacent to the dura over the right hemisphere. It was most marked at the base in the region about the mastoid, fading out toward the vault.

The patient made a good recovery from his operation and was somewhat relieved. His hydrocephalus still persisted, however, and he has again developed rhinorrhea.

Sections from the membrane removed at operation, which Dr. Naffziger was kind enough to forward for comparison, showed a thick layer of fibrous tissue, apparently of the same age throughout. In the stratum next the dura was a series of large, endothelium lined spaces filled with blood, resembling in general arrangement those seen in other cases. One of them, however, had at least two coats. They appeared to communicate with other blood vessels. There were several large, unencapsulated areas of hemorrhage throughout the sections. One of these surrounded a capillary and appeared to have resulted from its rupture. The neighboring tissues were infiltrated with fresh blood cells, perhaps from operative manipulation. The free surface of the membrane, toward the arachnoid, was comparatively avascular. On it there lay a small hematoma with beginning organization. A very thin fibrous layer covered by mesothelium overlay it. There were a moderate number of leukocytes of various sorts scattered through the membrane but no significant, localized collections of them. There was considerable pigment.

The classification of this case offers many difficulties. One must assume that the patient had sustained a fracture through the cribriform plate with the establishment of a rhinorrhea which temporarily drained his hydrocephalic ventricle. At the same time, a thin subdural hematoma was formed which became completely organized without cyst formation. The age of the fibrous membrane removed would correspond fairly well with the interval since his accident, but there appears to be a fresher hemorrhage on top of it, as if in this case the lesion were a progressive one. In only one other case in this series were multiple areas of hemorrhage observed.

CONCLUSIONS

1. An apparently insignificant trauma to the head may in certain individuals be followed, after a latent interval which varies from a few hours to months or even years, by symptoms of cortical irritation and of intracranial pressure due to a subdural hematoma. These cases are less rare than formerly supposed.

2. Clinically, after a period of headache, these patients show an extraordinary variability of symptoms, particularly in the intellectual sphere. They are prone to psychoses and often become irritable, indecent and unmanageable. Choked disk is apt to be present and a frontal lobe tumor suspected.

3. At operation or necropsy, a subdural hematoma will be found on one or both sides enclosed in a continuous membrane slightly adhering to the dura but not adherent to the arachnoid.

4. The enveloping membrane on the side toward the arachnoid is thin and covered with mesothelium. On the dural side, it is more dense and composed of organizing granulation tissue containing large mesothelium lined spaces containing blood and fibrin which appear to anastomose with each other and with the capillaries.

5. In this respect, the membrane of the traumatic hematoma seemingly differs from that of the commonly described pachymeningitis hemorrhagica interna, in which the thin-walled vessels are enormous, and no mesothelium lined spaces are seen. Such a pachymeningitic membrane may possibly give rise to hematomas, which symptomatically and in the gross resemble the posttraumatic variety.

6. In certain cases in which the onset of symptoms is delayed for only a few days after trauma, it is possible that they may be due to a hematoma formed immediately but which only announces its presence when the brain becomes edematous or congested.

7. In other cases the occurrence of late hemorrhages seems probable and may be due to the formation of communication between the mesothelium lined spaces and blood vessels, with rupture of one or the other.

8. The spontaneous pachymeningitis vasculosa may represent a chronic form of the same process. There is no satisfactory evidence that either form is inflammatory or neoplastic.

9. The term reactive pachymeningitis might be used to designate the peculiar process of organization that is evoked by the presence of blood clot or other collection of fibrin on the inner surface of the dura, and which may possibly give rise to secondary hemorrhages.

10. When the presence of a subdural hematoma is under suspicion, an exploratory craniodural puncture over the hemisphere will give the most reliable evidence of the presence or otherwise of the clot. This should be bilateral as the lesion is not infrequently bilateral. A lumbar puncture may show a faint xanthochromic fluid, but often the cerebrospinal fluid is negative.

11. If the diagnosis is verified, an osteoplastic resection with reflection of the dura and removal of the more or less organized clot as intact as possible is the procedure of choice, with subsequent painstaking hemostasis. The removal of the dura on the basis that it is a possible source of further hemorrhage is unnecessary.

12. Experience tends to show that there need be no fear of reformation of the clot but that cerebral edema is apt to occur in the brain released from its long pressure. It consequently may be advisable to combine the osteoplastic exploration with a subtemporal decompression.

13. Should edema occur, the use of hypertonic saline or the performance of lumbar puncture should perhaps be resorted to before reinvestigation of the wound, for in no case in the present series at least has the procedure been of use.

LOCALIZATION OF LUNG ABSCESS BY THE ROENTGEN RAY, WITH SPECIAL REFERENCE TO THE USE OF THE HIRTZ COMPASS

A PRELIMINARY REPORT *

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The practical value to the surgeon of the roentgen-ray localization of lesions depends on two essentials: the possibility of describing the position of the lesion in relation to landmarks, either natural ones or those artificially supplied by opaque markers, and the availability of such information to the surgeon working through the wound in his narrow aseptic field. The Hirtz Compass was originally designed to record this information in the case of foreign bodies, and to render it available to the surgeon with mechanical accuracy under aseptic conditions. The present paper is a preliminary report extending the use of the Hirtz Compass to the localization of cavities within the lung.

To any one with even slight experience in thoracic surgery the need of such a method of entering the cavity of a lung abscess with the exploring needle is evident. The large abscess rarely offers difficulty; but the small abscess, with or without subsidiary abscesses, often requires repeated insertion of the needle in many directions if the surgeon is guided only by physical signs and the findings obtained by the ordinary roentgenographic examinations. Under such guidance, the abscess may not be entered at all.

Even if such an unfortunate result does not occur, the trauma to the normal lung and the possibility of sowing infection broadcast make repeated blind needling undesirable. The use of the Hirtz Compass has enabled us to enter the cavity with assurance on the first introduction of the needle in three cases.

For those not familiar with the Hirtz Compass (Fig. 1), a brief description of its construction and the principle involved in its appli-

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cation, will be given. The Hirtz Compass is constructed entirely of metal. It consists of three movable horizontal arms which revolve in a horizontal plane about a fixed point. Each arm carries a vertical, adjustable leg; adjustable both as to position along the extent of the horizontal arms and as to height. The purpose of these horizontal arms and vertical legs is to form a framework

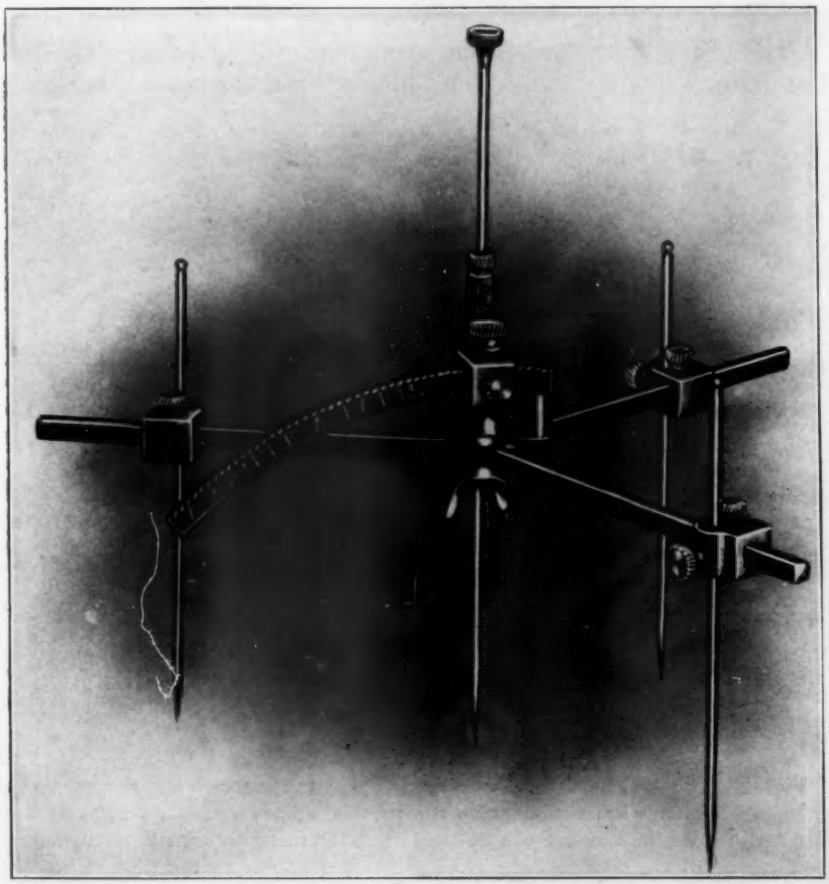

Fig. 1.—The Hirtz Compass, described originally for the localization of foreign bodies; a hollow needle is substituted for the indicator rod when used for lung abscess localization.

for the support of a pivoted curved arm which describes a meridian arc of a sphere; this is pivoted at the center or common attachment of the horizontal arms, and is movable about its pivot throughout the entire upper portion of the sphere. An adjustable slide carrying an indicator rod, held in the exact direction of the radius of the sphere, is movable along the meridian arc. Zero marks

are established on the vertical legs and on the indicator rod, measuring a distance equal to the radius of the sphere. Graduations are made on the vertical legs on either side of the zero mark in centimeters and half centimeters. With all the legs set at zero, the compass will stand in a horizontal position; and the indicator rod, introduced to its zero mark, will indicate a fixed point (the center of the sphere of which it is a radius) in whatever position it is placed along the meridian arc and wherever the arc itself is moved.

The principle involved in the application will be briefly described. The distance that the image of an opaque body moves on a recording

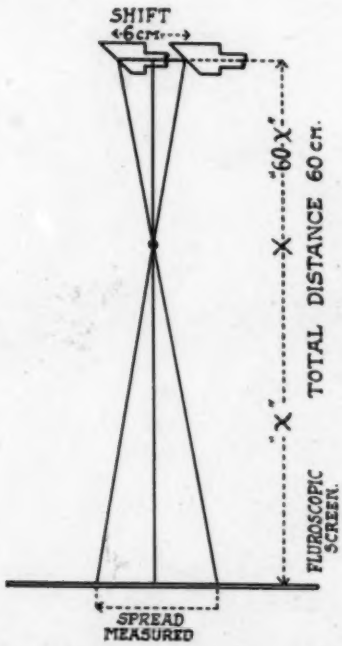

Fig. 2.—The distance that the image of an opaque body moves on a photographic film when the roentgen-ray tube is a known distance away and is displaced; a definite amount can be used in determining by simple triangulation the distance of the opaque body from the target and from the film.

surface (fluoroscopic screen or film), when the roentgen-ray tube is moved over a known distance, determines by simple triangulation the distance of the body from the target and from the screen (Fig. 2). The farther the body is situated from the fluoroscopic screen, the greater is the distance traversed by the image of the body on the movement of the tube. This method may be used in determining the respective distances of the cavity of the lung abscess and of three opaque markers placed in convenient locations on the skin around the presumed site of the abscess. With this information the difference in depth between

each skin marker and the abscess can be calculated. Now, theoretically, if the legs and indicator rod of the compass could be projected beneath the skin into the tissues, until the indicator rod passed into the abscess, the arms remaining in a horizontal plane, then it is quite evident that if the indicator rod were changed in position on the meridian arc of the sphere, the tip of the rod would accurately indicate the position of the abscess in any direction from which it was approached (Fig. 3, upper). If the plane of the abscess is arbitrarily accepted as the zero plane, then

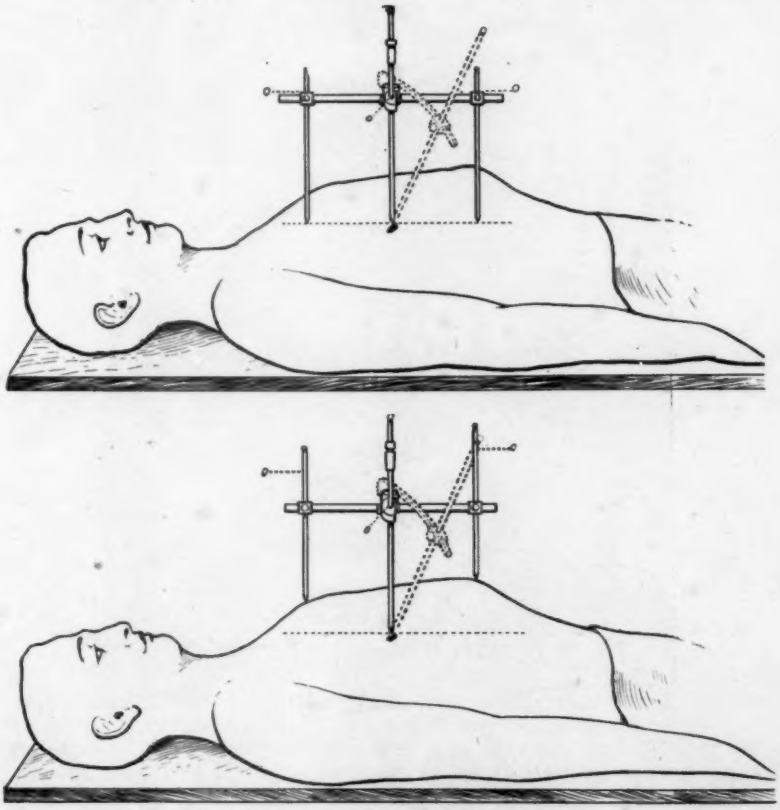

Fig. 3.—The Hirtz compass in use.

the difference in depth of the plane of the abscess, or zero plane, and of the plane of each individual skin marker will represent the distance which each adjustable leg would have to be introduced into the tissues or (in actual practice) the amount by which each leg will have to be shortened, in order to secure a horizontal plane for the compass while the legs rest on the skin markers (Fig. 3, lower). It is obvious, then, that with the instrument secured in this position, since the abscess was arbitrarily placed at the center of the meridian arc of the compass, the

indicator rod will point at any position directly to the abscess and if projected into the tissue will exactly enter the abscess. If obstructed by the skin surface, the depth of the abscess in that particular direction will be indicated by the distance which the indicator rod must yet be introduced in order to reach the zero mark. If a hollow needle is substituted for the indicator rod, an immediate aspiration of the pus will give assurance of the correctness of the calculation.

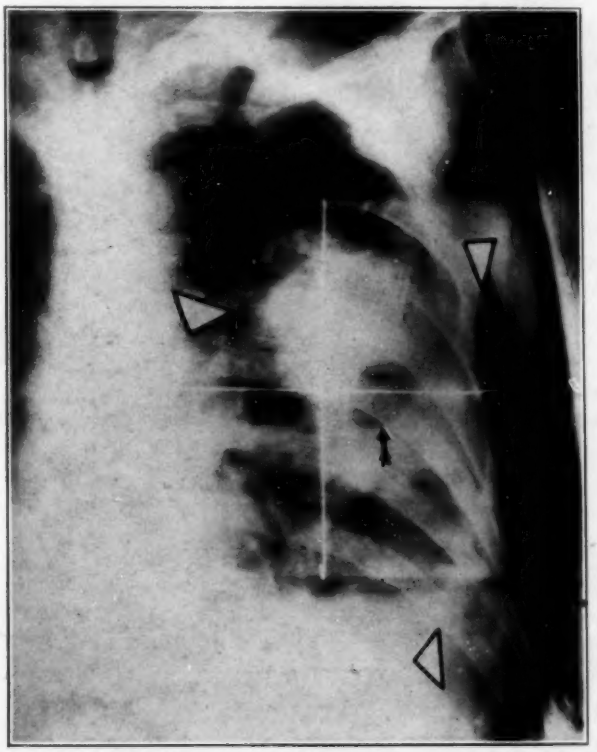

Fig. 4.—One of the two plates secured for the calculation of the setting for the Hirtz Compass. Note should be made of the three triangular opaque skin markers grouped around the abscess and the cross lines on the film which serve to center the films.

The advantages offered by this method may be briefly considered. Localization of this character aids the surgeon in determining the best means of approach for surgical drainage; the compass allows a wide selection for the operative field and will indicate the nearest point of the abscess to the skin surface. The method secures an accurate localization of the lesion, so that the abscess may be entered with the first introduction of the needle. The all-metal construction of the compass makes it easily sterilized, so that it may be utilized during the operation at any time to recheck the position of the abscess.

Some of the precautions to be observed, and the roentgenologic difficulties encountered in thus extending the use of the Hirtz Compass to the localization of lung abscesses, are worthy of brief consideration. In dealing with a lung abscess, it is obvious that we are dealing with a much less distinct image than is the case with a metallic foreign body. Since the accuracy of the method depends entirely on the clear visualization of the abscess walls in the two positions indicated by the shift of the roentgen-ray tube (Fig. 4), any indistinctness of the image of the abscess would render the examination valueless. In multiple small abscesses without definitely discernible outline, and in gangrenous conditions of the lung where no well defined wall can be seen, the method cannot be used.

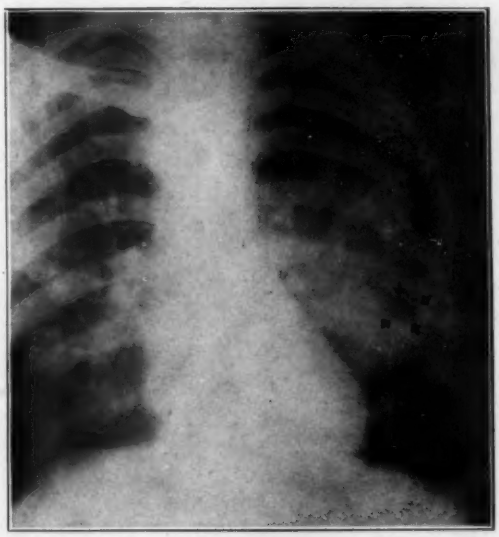

Fig. 5.—A small abscess cavity present in the left lower lung. The aspiration of pus at the first introduction of the needle was, in this case, a severe test of the method, as the actual cavity was probably no more than 2 cm. in diameter.

A number of attempts are often necessary in a favorable case before an examination suitable for localization is obtained. In chest roentgenography, a 60 cm. distance (the standard target-film distance for Hirtz Compass examinations) is not sufficient to secure the detail necessary in the roentgenogram. For this reason, it has been found advisable to increase this distance to 80 cm. The accompanying table of shift-depth ratios has been worked out on this basis. Since the abscess will appear clearest in its closest position to the roentgen-ray film and since the operative route must be, in general, at the point where the abscess approaches most closely to the skin surface, and furthermore, since the

patient must assume in the localization the position which he will assume on the operating table, it is clear that the most favorable position for operation, namely, with the skin surface nearest to the abscess, uppermost, is the least favorable position for Hirtz Compass localization, as the abscess is then farthest from the plate. This is the most serious difficulty encountered, but it is readily overcome by such an increase in the target-film distance. Care must be taken that the patient does not breathe and that both exposures are made in rapid succession during the same breath (the breath should be held neither in forced inspiration nor expiration, but in repose) since the accuracy of the calculation depends on a small variation in the position of the shadow of the

Shift-Depth Ratios

Focus plate distance, 80 cm.; Tube shift, 8 cm.

Spread	Height	Spread	Height	Spread	Height
0.10	0.99	1.75	14.36	3.40	23.86
0.15	1.47	1.80	14.70	3.45	24.11
0.20	1.95	1.85	15.08	3.50	24.35
0.25	2.42	1.90	15.35	3.55	24.59
0.30	2.89	1.95	15.68	3.60	24.82
0.35	3,35	2.00	16.00	3.65	25.06
0.40	3.81	2.05	16.32	3.70	25.30
0.45	4.26	2.10	16.63	3,75	25.53
0.50	4.71	2.15	16.94	3.80	25.77
0.55	5.15	2.20	17.25	3.85	26.00
0.60	5.58	2.25	17.56	3.90	26.22
0.65	6.01	2.30	17.86	3.95	26.44
0.70	6.44	2.35	18.16	4.00	26.66
0.75	6.86	2.40	18.46	4.05	26.89
0.80	7.27	2.45	18.75	4.10	27.11
0.85	7.68	2.50	19.05	4.15	27.33
0.90	8.09	2.55	19.34	4.20	27.54
0.95	8.49	2.60	19.62	4.25	27.75
1.00	8.89	2.65	19.91	4.30	27.97
1.05	9.28	2.70	20.19	4.35	28.18
1.10	9.67	2.75	20.46	4.40	28.39
1.15	10.06	2.80	20.74	4.45	28.60
1.20	10.44	2.85	21.01	4.50	28.80
1.25	10.81	2.90	21.28	4.55	29.00
1.30	11.18	2.95	21.55	4.60	29.21
1.35	11.55	3.00	21.82	4.65	29.41
1.40	11.91	3.05	22.08	4.70	29.61
1.45	12 28	3.10	22.34	4.75	29.80
1.50	12.63	3.15	22.60	4.80	30.00
1.55	12.98	3.20	22.86	4.85	30.19
1.60	13.33	3.25	23.11	4.90	30.39
1.65	13.68	3.30	23.36	4.95	30.58
1.70	14.02	3.35	23.61	5.00	30.77

abscess with the two positions of the roentgen-ray tube. The skin markers should be placed so as to present a good footing for the compass, and their position should be jointly decided by the surgeon and the roentgenologist. Their position should be permanently recorded on the skin by scratching the skin at these points. We have not found a satisfactory material for marking the skin which will resist the operative preparation of the patient. Irregularities in the margins of the abscess cavity are of no consequence since the roentgen ray gives a composite view of an indefinite number of sections through the abscess and since, to provide the maximum amount of leeway, the center point of the abscess is the one selected for localization. It is possible that the

abscess may not show as well in one position as it does in another, so that several attempts may be necessary before a dependable localization can be made. All attempts at localization should be checked by a second examination before the patient is sent to operation. No attempt should be made in localization of lung abscesses until the roentgenologist is thoroughly familiar with the application of the instrument to the localization of foreign bodies.[1]

Besides the roentgenologic difficulties described, we have encountered several minor surgical pitfalls which, with care, can be largely avoided. We detail them so that the surgeon and roentgenologist may be on the alert to avoid them. Although they may seem formidable as they are described, yet with ordinary care in minute details of technic, the difficulties will be successfully overcome. They are:

1. The difficulty in placing the foot points of the compass so that they will be near enough to the site of the incision to be easily included in the sterile field, and at the same time far enough away so that the mass distention of the tissues with local anesthetic solution will not disturb their various levels.

2. The necessity, at times, of more than one roentgenographic localization. If the abscess is localized by the Hirtz Compass to the entire convenience of the roentgenographer, it will often be found that the later election of the operative site on the basis of the compass localization will prevent the use of the points determined; since either the patient cannot be comfortable on the table in the position chosen, or the operative site is found to lie too near the table for convenient manipulation. The localization must then be repeated with the patient in the exact position that he will assume on the operating table. This difficulty is minimized, as we have already remarked, by close co-operation between surgeon and roentgenographer throughout the procedure.

3. The errors introduced as the direct result of operative treatment in one stage and two stage procedures. If the pleura is not adherent and the first stage is employed to insure adhesion, the operation results in swelling of the thoracic wall in the neighborhood of the incision and an inflammatory process in the pleura and adjacent lung which may displace the skin markers relative to the abscess or the abscess relative to the skin markers, or both. When an obliterated pleura is found at the first operation, the slight retraction of the lung incident to rib resection may permit the abscess to draw slightly away from the chest wall. The permissible margin of error is so great, however, even when one is dealing with a relatively small abscess, that the slight retraction of the abscess incident to the removal of a small segment of a rib

1. Full detail of the method of application of the Hirtz Compass is given in the United States Army Roentgen-Ray Manual, pp. 241-266.

would hardly be sufficient to invalidate the localization. In the localization, the center of the cavity is localized so that there is a permissible margin of error in all directions equal to the radius of the cavity. If the abscess cavity is extremely small, however, it is easy to conceive how the sum of these errors might easily result in complete failure to enter the abscess with the pointer. When the abscess is small and a two-stage operation is necessary, a second localization must be made, even though under the technically difficult condition presented by a fresh wound in the roentgenographic zone.

4. It is possible that the pus may be too thick to be withdrawn through the very long, small gage needle that we have substituted for the indicator rod. If a tap should prove negative, there can be no objection to the withdrawal of the needle for the purpose of examining its tip for pus or necrotic material, with the meridian arc and the needle collar left clamped in the original position. The needle can then be reintroduced through the same path to the abscess. This is, of course, an advantage of the ordinary exploratory aspiration in which the same accident may occur.

5. The cumbersomeness of the apparatus and technic. We consider this, however, a disadvantage of no weight, if we can repeat indefinitely our successful exploration of the lung with such accuracy. Further experience undoubtedly will show us methods of simplification.

We have found several accessories to the original apparatus of value in this special application of the compass. The replacement of the indicating rod by a hollow needle with a supporting collar permits the aspiration of pus directly through the needle indicator rod. Rubber tips slipped over the feet of the compass eliminate much of the discomfort to the patient from pressure of the sharp points during localization and operation. Although the short meridian arc furnished with the apparatus has been used and gives a wide selection for the field of operation, the longer arc would greatly enlarge this field and would be of especial advantage in cases in which the abscess presented near the axillary line.

We have employed the Hirtz Compass on the operating table in three cases of lung abscess; two of these were drained by one of us; the third patient was operated on by Dr. Louis Rassieur, who furnished the clinical data.

REPORT OF CASES

CASE 1.—A man, white, aged 31, was under observation and medical treatment at the St. Louis City Hospital for three months prior to operation (Fig. 5). He complained on admission of pain in the chest; hemoptysis; foul bronchorrhea, and loss of weight. All these symptoms followed a "cold" which he had had three months previously. The roentgenographic examination revealed a small abscess cavity in the middle portion of the left lower lobe.

March 5, 1923, the parietal pleura was exposed under a local anesthetic and, by sutures and packing, adhesions between the parietal and visceral pleura were encouraged. A week later the abscess was drained by the actual cautery without an anesthetic. In this case, the operative site was chosen under direct guidance of the Hirtz Compass. At the second stage, the Hirtz Compass was again used to guide the exploring needle. In this instance, the swelling about the wound, plus the slight dropping away of the lung from the chest wall had so separated the planes of markers and abscess that the abscess was not entered with the indicator at the zero mark. It was necessary to push it in the same direction about a centimeter before pus was encountered. The aspiration of pus at the first introduction of the needle was, in this case, a severe test of the method, as the actual abscess cavity was probably no more than 2 cm. in diameter. The slight error in depth could have been avoided by the precautions already discussed had it not been our first attempt with the method.

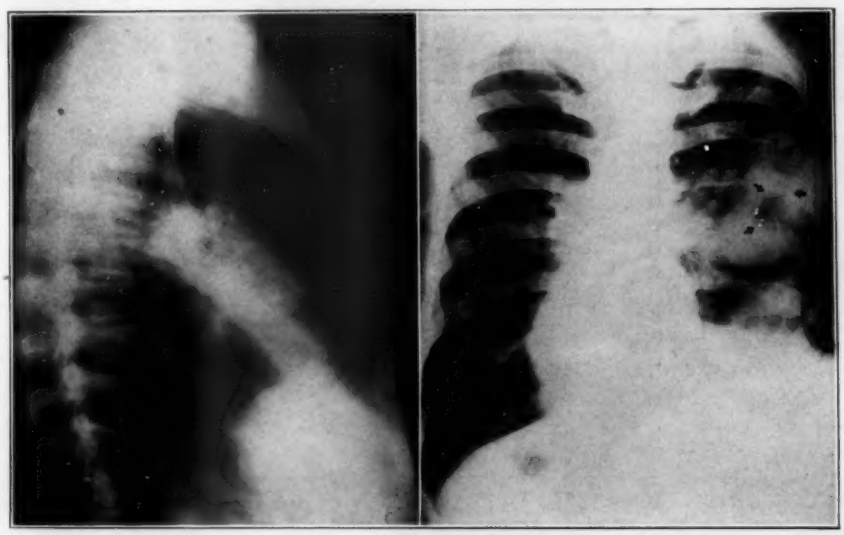

Fig. 6.—The abscess presumably was the result of the rupture into the lung of a postappendicitis subphrenic abscess. In the lateral view it was seen that the infection traversed practically the entire lower lobe. In using the Hirtz Compass we were impressed, in this case, by the ease with which the cavity was entered from the lateral direction, although the compass skin markers were placed entirely on the anterior surface of the chest.

This patient gradually improved in general and local condition and left the hospital in July, 1923, without symptoms. His wound was almost healed and his roentgenogram showed almost complete return to normal. Reexamination June 5, 1924, showed complete recovery, both clinically and roentgenographically.

Case 2.—A man, white, aged 33 years, presented a history of sixteen months' duration. The abscess presumably was the result of the rupture into the lung of a postappendicitis subphrenic abscess (Fig. 6). It traversed practically the entire right lower lobe. Months of medical treatment had been given without improvement and he presented the usual distressing picture of emaciation, foul

bronchorrhea, cough and daily pyrexia. On Jan. 9, 1924, this abscess was drained under local anesthesia at the St. Louis City Hospital. Three inches (7.6 cm.) of the fourth and fifth ribs on the right side in the anterior axillary line were resected and the operation was completed in one stage with the actual cautery. In using the Hirtz Compass we were impressed in this case by the ease with which the cavity was entered from the lateral direction, although the compass markers were placed entirely on the anterior surface of the chest. The cavity was rather large, so that entering it at the first introduction of the needle was a test of the applicability of the method to the lateral approach rather than a test of its accuracy in general.

This patient was promptly relieved of his symptoms, gained 30 pounds (13.6 kg.) in weight and four months after operation was able to undertake

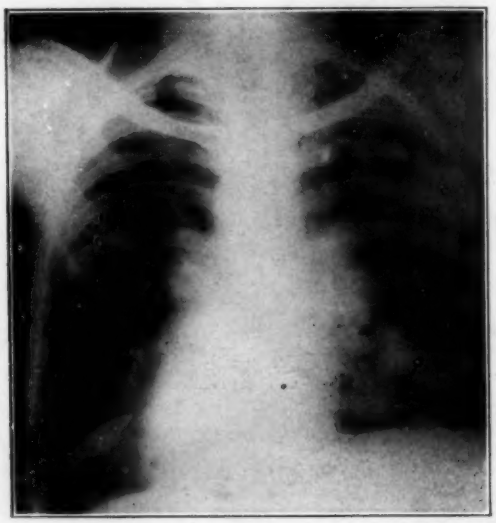

Fig. 7.—A moderately large abscess cavity, partially filled with fluid in the right lower lobe. This represents the type of abscess best suited to Hirtz Compass localization.

light work. The cavity was clean, but was still several centimeters in diameter and contained small bronchial fistulas.

Case 3.—A man, white, aged 36, entered St. Mary's Hospital complaining of cough, pain in the chest, dyspnea and a loss of 23 pounds (10.4 kg.) in weight over a period of eleven months. The onset followed exposure to cold and wet without a history of preceding illness.

The clinical picture suggested pulmonary tuberculosis. On roentgenographic examination a moderately large abscess cavity, partially filled with fluid, was found in the right lower lobe (Fig. 7).

The bronchoscopic examination confirmed the diagnosis of lung abscess. The abscess was drained in one stage by Dr. Louis Rassieur. At operation, the indicator needle of the Hirtz Compass passed readily into the abscess cavity. This type of abscess with well-defined walls represents the most favorable type for localization.

CONCLUSIONS

1. By the employment of the Hirtz Compass for localization in three cases of lung abscess, we have been enabled on the operating table to enter the cavity of the abscess with the exploring needle at the first attempt.

2. At least one of these cases (Case 1) offered a severe test of the accuracy of the method.

3. We can describe certain sources of error to be avoided, but our experience is still much too limited to assure us that these are the most serious sources of error.

4. The use of the Hirtz Compass for localization appears to us to offer promise in increasing the accuracy of surgical attack on those cavities within the lung the walls of which present clear-cut roentgenographic shadows.

ABSTRACT OF DISCUSSION

Dr. Edward S. Blaine, Chicago: The Hirtz Compass is an instrument of 100 per cent. accuracy for the purpose for which it is intended. In applying it to the human body, as Dr. Sante has found, a disturbing factor is the movability of the skin on which the legs of the instrument must necessarily be placed. During the war I taught a number of doctors the use of this instrument in locating foreign bodies on wooden models, and it was so simple and easy that one wondered why it was not universally used. It was 100 per cent. accurate every time. But applying it to the human body, in actual practice, it has been found that we do not get 100 per cent. accuracy. This is probably due to the lack of a large enough experience with the human body. It is evidence of genuine skill on the part of the operator if he succeeds in entering a small abscess, as Dr. Sante has done. He is to be complimented on this application of the Hirtz Compass, which is truly a very ingenious method in the locating of the particular lesion under discussion. We are all familiar with the too numerous stab wounds inflicted in attempting to reach a lung abscess with the needle. As to the setting of the compass, it will be a difficult matter at best to select the three points necessary for the three legs to rest on. Dr. Sante's diagrams are very assuring, and almost alluring, but it is a different matter to pick the proper points for this compass so that it will not tip over when left to stand alone. In some of the cases, one leg will seem to have to stand on the side of the chest wall. I wonder how Dr. Sante gets the third leg placed so that it will not slip off. The two legs on the chest (with the patient supine) are firmly placed, but this lateral leg is not. It was also found that in the various movements of the patient between the roentgen-ray localization and the operative work, these leg points were shifted or even lost. Dr. Sante has referred to the difficulty of getting a skin marking material which will withstand the sterilization procedure. In spite of these difficulties, which can be overcome, this interesting device will certainly aid the operator to enter directly into these abscesses.

Dr. W. H. Stewart, New York: The accurate localization of abscess of the lung with the roentgen ray is of vast importance. It must be done by the most simple means possible; the surgeon does not want to be bothered with a method that presents an extensive mathematical problem. For many years I

have depended on fluoroscopy, reinforced by stereoroentgenograms, with skin markers and good lateral films in the prone position, the tube in front and the plate behind. In a vast majority of cases of abscess of the lung, this technic will successively give the required information. Occasionally, however, it will not. One must not forget that when making such a localization it is necessary for the roentgenologist to place the patient in the exact position he is to be placed in when the surgeon has him on the operating table ready for the incision. The necessity of recording the phase of the respiration when the localization is made is of great importance. It is astonishing to note the amount of respiration excursion an abscess has when it is not caught by pleural adhesions. It is also important to localize it by relations and measurements to certain bony land marks. Skin marks are readily displaced in the extensive flaps necessary in chest surgery.

I have always felt that the ideal method of localization is by means of the double fluoroscope, which is used so successfully by Dr. Willis F. Manges in the localization of foreign bodies in the lung. Of course, objections to this procedure often occur, as many surgeons prefer to depend on what information one can give them by more simple measures. If Dr. Sante can overcome all these difficulties with the Hirtz Compass he has given us great help. It is to be regretted that the use of this instrument is limited to those cases in which the cavity is clearly outlined. The associated lung condition surrounding the abscess cavity is so likely to produce a certain fuzziness in the roentgenogram that clear detail, unless in very chronic cases, is often lacking.

Dr. David R. Bowen, Philadelphia: In 1917 I studied the Hirtz Compass carefully and understood it very well. Last winter I had occasion to use it for class teaching and, to my astonishment, had much difficulty in getting a proper conception of its use.

Do not order a Hirtz Compass and tell the surgeon that you will localize this abscess for him "next Friday." It will take time to learn how to use it; you must study the compass on the model, study it on stilts, and thoroughly master it before making promises. Its use is fairly simple after it is learned, but it is very difficult in the beginning.

Dr. P. M. Hickey, Ann Arbor, Mich.: It is well to recognize the limitations of the Hirtz Compass. It is particularly applicable when one has a definite framework on which to support the legs of the compass, as to localize foreign bodies in the head. The unyielding surface of the vaulted skull makes it possible, for the legs of the compass always to occupy the same relation to the foreign body. This method is very exact in chest cases if one selects the bony framework as a rest for the legs of the compass. It will then be quite accurate. If one attempts to use the Hirtz Compass on yielding structures, as in the localization of foreign bodies in the thigh, great difficulty will be met in preserving the relative position of the points of support. This paper reminds me of the work of localization of foreign bodies of the chest which I saw done by Dr. Pette Del ve Leon of Paris during the war. He was able to localize fluoroscopically pieces of shrapnel in the lung so accurately that through a slight skin incision he passed a blunt nosed forceps through the skin, pleura and lung, pushed aside the great vessels, and picked up pieces of shrapnel no larger than a finger nail. It is evident that the fluoroscopic method is of the greatest importance in the localization of these foreign bodies in the lungs.

Dr. E. P. Lehman, St. Louis: Dr. Sante has asked me to answer one of the several questions that have been brought forward, namely, Dr. Stewart's

suggestion that it is difficult to preserve the skin marks throughout an extensive surgical prcedure. We have not found in the three cases in which we have tried the method that we have lost the marks. If a position could once be found for them, they were available throughout the operation. It may be necessary to limit the incision at first to a certain extent to preserve the marks, at least until the cavity of the abscess has been found by the exploring needle. Thereafter, there is no further need for the marks. I want to emphasize three points from the operator's point of view, on the basis of our extremely limited experience with the method. The first is that the difficulty in the method is entirely the roentgenographer's. When the machine has once been set on the base points, with the patient in the determined position, then on the operating table the finding of the abscess is the simplest thing in the world. This applies to either the anterior, posterior or lateral approach. The second point is the very great accuracy with which the exploring needle enters the cavity. The third point is the advantage of such accuracy to the patient as well as to the surgeon in eliminating extremely undesirable multiple exploratory punctures through which unnecessary trauma is done to the lung and pleura and by which infection may be spread to a portion of the lung not already involved.

Dr. L. R. Sante, St. Louis: I want to emphasize the point Dr. Stewart made, that this is an attempt to get over a serious difficulty. We have, as I said, in three cases been able to enter the abscess cavity on the first insertion of the needle. We have substituted for the indicator rod of the Hirtz Compass a hollow needle with proper attachment so that a syringe can be attached and pus aspirated directly through the needle. It requires an extra collar and bushing to hold the needle absolutely in line. One thing I would like to point out: the adaptation of this instrument to lung abscesses is not exactly the same as its use in localizing foreign bodies. In the case of a foreign body, you rarely have anything as large as an abscess to localize. Rarely do you have an abscess as small as the first one we showed here, 2 c.c. in diameter; they are nearly always somewhat larger than that. In order to make this source of error give us the best chance to strike the abscess, we localize the center of the abscess shadow.

I should like to emphasize the applicability of the method to one definite, clear cut type of case, that in which the abscess is more or less well defined and the walls can be seen clearly in both positions of the roentgen-ray tube. The difficulties with the placing of the instrument and the foot marks can be overcome by increasing the length of the arc; the supporting framework can be up on top of the patient and the point of approach can be made laterally. I think this will be found to be a great aid in establishing the stability of the compass. Likewise, the compass can be held in position even if it is not horizontal and will accurately localize the abscess. This is a method which we hope will be of value in cases in which the abscess cavity is clearly defined and can be seen clearly. We think that with the flouroscopic setting of the compass, one of the methods described in the army manual, we may be able greatly to simplify the technic, and we are going to attempt to do this on some tuberculosis cavity, and come to some definite conclusion in this way.

THE REMOTE SEQUELAE OF RECTAL IMPLANTATION OF THE URETERS FOR EXSTROPHY*

FINDINGS AT NECROPSY FOURTEEN YEARS AFTER BERGENHEM OPERATION

DE WAYNE G. RICHEY, M.D.

PITTSBURGH

Although exstrophy of the urinary bladder is a rare deformity, occurring once or twice in every 50,000 births, it is a comparatively frequent form of the more formidable anomalies involving the genito-urinary tract. Briefly stated, bladder exstrophy consists of a congenital malformation in which, in the course of fetal development, there is a failure of union, anteriorly, between the two lateral halves of the urinary bladder and everything in front of it, including the abdominal wall, the pubic symphysis and the urethral roof. The space resulting is filled in by the posterior wall of the bladder and the urethral floor. In other words, it is a state of extreme epispadias.

The resulting condition is deplorable, "the victims being constantly wretched, offensive to themselves and their associates, and burdensome to those on whom they must depend." In addition, the male, who is affected at least three times more frequently than the female, has a rudimentary penis and undeveloped testes, necessitating a life of sexual inactivity. Naturally, inexhaustible efforts have been expended in the "laudable endeavor to correct such wretchedness and reclaim such sufferers to a comfortable existence, although nature has a compassion for many of these unfortunates, frequently shortening their expectancy of life, so that about two-thirds of all exstrophics die before reaching the age of twenty, usually of renal infection."

The results of the many ingenious procedures devised for the relief of these patients were highly unsatisfactory until 1892, when Maydl [1] first implanted the trigone of bladder with distal ends of the ureters into the sigmoid flexure. Two years later, Bergenhem [2] implanted, extraperitoneally, the ureters with a roset of bladder attached by separate openings into the rectum, a procedure that was destined to reduce the immediate operative mortality from 28 to 10 per cent. by lessening

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* From the Departments of Surgery and Pathology, Mercy Hospital.

1. Maydl: Concerning the Radical Treatment of Exstrophy of Bladder, Wien. med. Wchnschr. **44**:25, 1169, 1209, 1256, 1297, 1894; ibid. **46**:28, 1896.

2. Bergenhem: Exstrophy of Bladder with Implantation of Ureters into Rectum, Eira **19**:265, 268, 1895.

the chances of peritonitis and kinking of the ureters. Basically, this operation fulfilled every requirement of trigonal implantation by preserving the blood supply and guarding the ureteral orifices, which appear to be protected by a valvular rather than a sphincteric arrangement.

In 1909, Buchanan [3] published an exhaustive paper on the remote results of implantation of the ureters into the large bowel for exstrophy. He was able to collect cases of ninety-eight patients who had survived operation, including one of his own, and he referred in an addendum to a second personal case. Of these ninety-eight patients, eleven died subsequently of renal infection, while sixty-four were well at the time of publication, thirteen were well within one year, twenty-six between one and three years, ten between three and six years and fifteen between six and twelve years after operation. Arumugam [4] reported the cases of two boys who had survived extraperitoneal transplantation of the ureters for four and four and a half years, respectively, and mentioned two additional instances, in which the patients were living fourteen and fifteen years afer similar operations. Later, Mayo,[5] in emphasizing the advisability of tubularizing the ureteral entrance into the rectal wall, cited the outcome of thirty-seven exstrophics, twenty-two of whom were operated on, six by plastic methods, three by the Maydl method and thirteen by the "indirection" method. Four of these thirteen died within three years after operation, only one, however, being an operative death and the other three succumbing to an intercurrent disease. Demel [6] gave the mortality in 400 Maydl operations as 41 per cent. in children under 5 years of age, 18 per cent. in those between 6 and 10 years of age and from 50 to 72 per cent. in persons over 20 years of age. Mugniéry [7] reported good remote results in 60 per cent. of the patients operated on by the Maydl technic, and mentioned five from Josserand's clinic who had survived from twelve to twenty-two years after operation without any evidence of renal infection. Many sporadic allusions to persons who have lived for varying periods of time after one operation or another can be found in the literature. One of the longest of

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3. Buchanan: Remote Results of Implantation of the Ureters into the Bowel for Exstrophy, Surg., Gynec. & Obst. **8**:146 (Feb.) 1909.

4. Arumgam: Extraperitoneal Transplantation of the Ureters into the Rectum, Brit. M. J. **1**:754 (April 1) 1911; Extroversion of Bladder Treated by Extraperitoneal Transplantation of Ureters into Rectum, Brit. M. J. **1**:966 (May 2) 1914.

5. Mayo, C. H.: Exstrophy of Bladder and Its Treatment, J. A. M. A. **69**: 2079 (Dec. 22) 1917.

6. Demel, R.: Results of Operative Treatment of Exstrophy of Bladder, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **33**:533, 1921.

7. Mugniéry, E.: Remote Results of Maydl's Operation, Lyon chir. **18**:48 (July-Aug.) 1921.

these has been chronicled by Winslow,[8] who performed a modified Wood operation on a girl aged 6. Since the operation she had borne four children and was healthy at the age of 36, although she had a prolapsed uterus and a constant incontinence of urine.

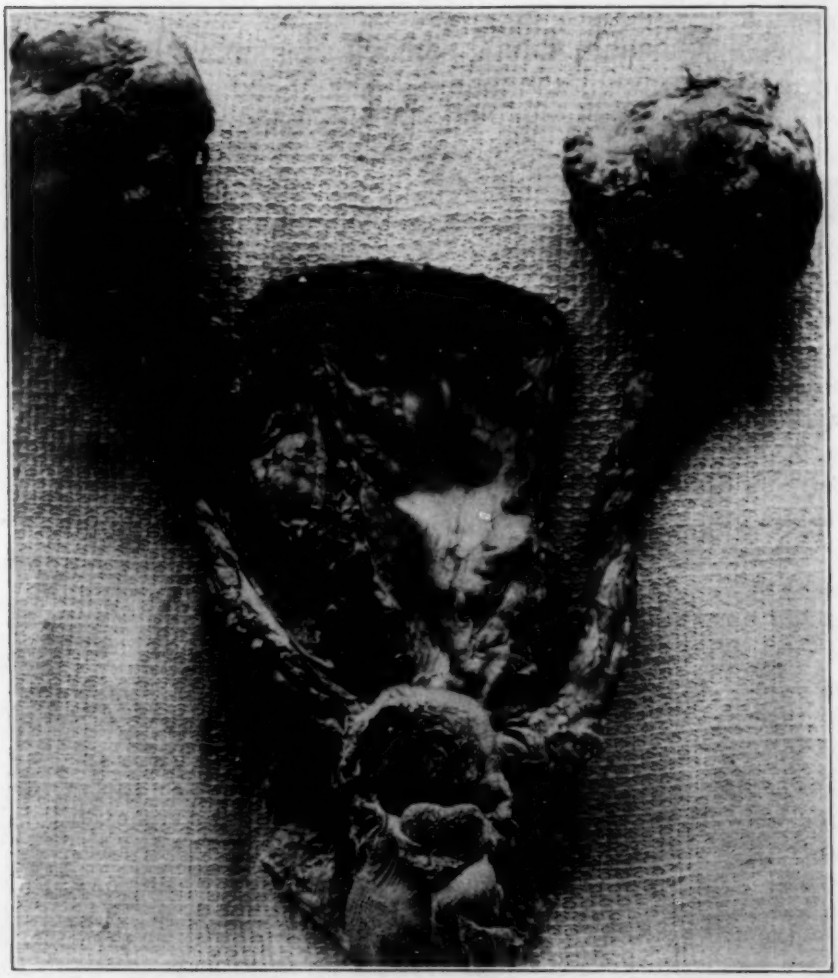

Fig. 1.—Ventral view, showing the disparity in size of the kidneys; the tortuous, thick-walled, dilated ureters; the fibrosis at the site of rectal implantation of ureters; the absence of bladder and the atrophic penis, directed upward in the depression between the united ends of the pubic bones.

It is not the object of this communication to discuss the relative merits of the various operations designed to correct this deformity, nor

8. Winslow: Report of Case of Exstrophy of Bladder Operated on Nearly Thirty Years Ago, Surg., Gynec. & Obst. **22**:350 (March) 1916.

can we add anything to our knowledge of its embryologic aspect, but as the case about to be reported is the same one to which Buchanan [3] referred in the addendum to his original article and, inasmuch as it is a fulfillment of his prophecy that "further reports are necessary in later years to confirm this favorable view, for some of these patients may yet die of renal disease," it is deemed worthy of record.

REPORT OF CASE

Operation.—Dec. 15, 1908, Dr. Buchanan performed the Bergenhem operation on a boy aged 3 years. Soon after the operation, the boy was able to retain his urine for as long as four hours. Although he was quite comfortable and was able to attend school, he was tardy in development. Nothing of a serious nature happened until October, 1920, twelve years later, when he presented himself with a left-sided pyonephrosis and perinephric abscess, which was drained. In May, 1922, almost fourteen years after the rectal implantation of the ureters, he returned again with a similar lesion on the right side. This, too, was drained and the patient died four days later.

Necropsy.—At necropsy, although the boy was over 16, he did not appear to be more than 6. The testes were atrophic and lay high in the inguinal canals. The penis was rudimentary and was directed upwards to rest in a depression caused by the failure of the pubic bones to unite mesially. Here a mass of scar tissue indicated the site of the previous operative excoriation of the floor of the exstrophied bladder. There was a complete epispadias of the penis, no vestige of a roof being present. The umbilicus was absent. In addition to the marked changes in the urinary apparatus, there was a recent verrucose endocarditis, involving the tricuspid, aortic and mitral valve leaflets, and a septic infarct of spleen.

The genito-urinary viscera were removed en masse, along with the attached sigmoid, rectum and anus, in order to ascertain the status of the former transplantation of the ureters into the rectum. The left kidney was small and globular, measuring 6 by 4.5 by 2.5 cm. It was embedded in dense white scar tissue, which was particularly abundant around the pelvis. The thickened, fibrous capsule could not be separated from the underlying renal tissue, which had become so involved in a destructive suppurative process that no normal landmarks could be recognized in it. In the midportion of the kidney, the calices were dilated and cystic, extending to the capsule. They contained a thick, reddish yellow purulent material and were lined with a rough, glassy pyogenic membrane of variegated colors. These cystic cavities were surrounded by dense deposits of white, fibrous, connective tissue in which yellow areas of desiccated pus and fat occurred. There was virtually no normal renal tissue remaining and the pelvis had been practically obliterated. The left ureter was 18 cm. in length. It was greatly enlarged and tortuous, and the surface was covered by many dense fibrous adhesions. At the lower end, where it had been implanted, extraperitoneally, into the rectal wall, the ureter was embedded in a mass of scar tissue. Along its entire course, the wall was thickened, rigid, tough and lacking in resiliency. The dilated lumen, which was occluded at the ureteropelvic junction, contained a brick red, mushy, foul smelling purulent material. When this was washed out and the lumen filled with water, it was found to measure 1.5 cm. in diameter, except at a point 1 cm. above its insertion into the rectum, where the caliber suddenly diminished to 5 mm. owing to the mural

fibrosis and the periureteral scar tissue. The mucosa was thick, red and velvety, and the longitudinal rugae stood out prominently.

Although the right kidney was relatively small, it was considerably larger than the left kidney, measuring 8 by 5.5 by 4.3 cm. On the thickened capsule were many fibrous adhesions, and on the posterior surface in the lower half was a heavy, yellow, friable pyogenic membrane, representing the wall of a recent

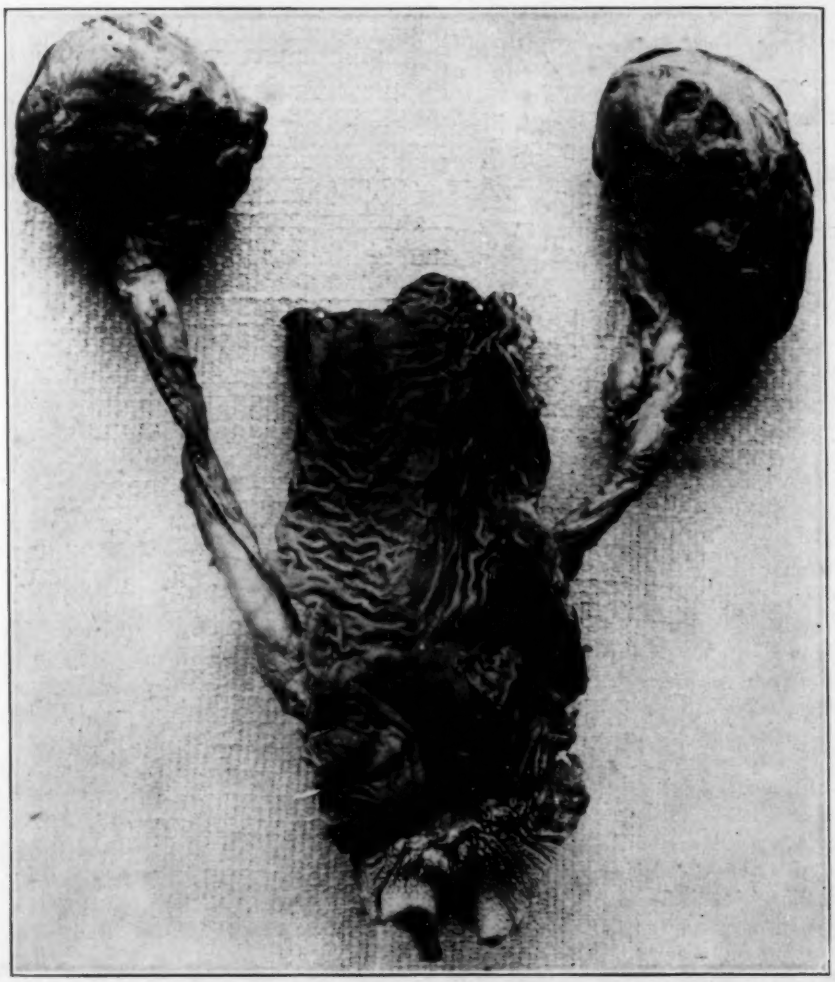

Fig. 2.—Dorsal view, showing the old scar tissue around the left kidney; a portion of the wall of the recent right perinephric abscess; the compensatory pouching of the rectum, and the orifices of the ureters in the anterior rectal wall.

perinephric abscess which had been opened at operation. The capsule peeled readily from the subjacent cortex, revealing a moderate amount of recognizable renal tissue. The line of demarcation between the cortex and medulla was poorly defined, and the cut surface was streaked by cords of white fibrous con-

nective tissue and deposits of yellow pus, which imparted a mottled appearance to the organ. The greatest damage was encountered in the lower half, where the pelvis was dilated and filled with a large stag-horn, hard, white calculus, measuring 3 by 2 by 1 cm. This concretion sent short, arborescent projections into the dilated calices. Four of these cystic calices in the lower half and one in the upper half varied in diameter from 1 to 3 cm. They harbored a thick, mushy, purulent material, which clung to the roughly corrugated, shiny, injected, thickened lining. The cut ends of the blood vessels stood out prominently, as a result of the mural thickening. The right ureter presented a picture not unlike that observed in the left. It, too, was thick and tortuous, measuring 1.6 cm. in diameter when filled with water. There were many fibrous adhesions around the ureter, notably at its lower end, where the wall attained a thickness of 7 mm. in contrast to 3 mm. above. The contents of the ureteral lumen consisted of a thick, odoriferous purulent material, simulating that found in the renal pelvis and calices. The ureteral mucosa was swollen and congested, exhibiting prominent velvety longitudinal folds. Where the ureter had been implanted into the rectal wall, its lumen was very small, able to accommodate only a small probe.

The anal orifice was small, and the sphincter muscles were well developed. Just above the anus the rectum was dilated, having a fusiform shape when it was filled with water. This pouch measured 8.5 by 8.0 cm. a distance of 6 cm. above the anus. Proximally to this dilatation, the large intestine was of smaller caliber and its wall was thinner and more elastic. In the dilated portion small, sessile diverticula occurred between the longitudinal muscle bands. The rectal mucosa was healthy in appearance. Into the anterior wall of the rectum, at a point 2.7 cm. proximal to the anal ring, the left ureter had been implanted. The right ureter also had been inserted through the anterior wall, 3.5 cm. proximal to the ring and 1 cm. to the right of the left ureteral orifice. Both these openings were round and small, and were well guarded by the rectal rugae. The mucous membrane was everywhere intact. Whether or not the rectal wall had developed a sphincter-like action around the transplanted ureters could not be ascertained. In the intact specimen, when the rectum was filled with water, the ureters were hydrostatically dilated at once by virtue of a ready communication between the rectal and ureteral lumina.

Microscopically, sections of the left kidney showed a very diffuse and extensive inflammation throughout the tissue. In places, the normal glomerular structures were entirely absent or completely hyalinized, and frequently the tubules were compressed and atrophic. Only occasionally could be seen a small area of relatively normal renal cortical tissue. Throughout the infiltrated portions were dense masses of lymphoid and plasma cells. Polymorphonuclear leukocytes were not present in any appreciable numbers. The hyalinized glomeruli were localized almost entirely to the inflammatory portions of the section, where the blood vessels also showed a marked thickening of the walls, involving an intimal hyperplasia and hyaline thickening of the media. In those regions near the pelvis, the inflammatory process was most intense, and here many polymorphonuclear leukocytes occurred. One section, taken through the pelvis, showed that the normal lining was gone, having been replaced by a thick granulation tissue, on the surface of which was a considerable degree of necrosis. Resting on the granulation tissue, in another place, was a thick layer of stratified squamous epithelium.

Microscopic section of the right kidney showed a very thick fibrous capsule, well infiltrated by inflammatory cells, including polymorphonuclear leukocytes

and lymphocytes and plasma cells. The cortical substance was much less involved than in the left kidney. Generally, the glomeruli had a healthier appearance. Here and there in the interstitial tissues, collections of inflammatory cells were encountered. There was a moderate degree of swelling of the tubular epithelium, with some tendency to desquamation. In the collecting tubules, some inflammatory cells occupied the lumina, although this was less marked than on the left side. Another section indicated considerably more inflammation in the cortical area, especially in the interstitial tissues, with a resultant disappearance of tubules. An occasional glomerulus revealed partial hyalinization with definite thickening of Bowman's capsule.

Microscopic sections of the ureters showed a thick, rather edematous wall. The normal mucosa had become replaced by a thick layer of loose granulation tissue, portions of which were necrotic. The muscle bundles were widely

Fig. 3.—Kidneys in longitudinal section, showing atrophy and fibrosis with cystic calices and obliterated pelvis of the left kidney; the more recent pyonephrotic process and staghorn calculus in pelvis and calices of right kidney.

separated by a loosely edematous tissue and were extensively infiltrated by lymphocytes, plasma cells and polymorphonuclear leukocytes. The inflammatory process had extended into the serosa, and around the perivascular spaces of the larger vessels were sizable collections of lymphoid cells. The most intense inflammatory reaction, however, occurred in the inner layers.

Bacteriologically, a paracolon bacillus, giving the tinctorial reactions of *Morgan's bacillus I,* was isolated from the perinephric abscess, from both kidneys and ureters, and from the heart's blood.

The exact pathways by which infection ascends from the lower urinary tract to the kidneys is as yet an open question, although the

problem has been subjected to exhaustive experimental observations by many investigators. That renal infection does occur following the rectal implantation of the ureters in dogs is well known from the experiments of Martin,[9] who described an ingenious operation which he had performed on 37 dogs, 4 of which lived for indefinite periods after operation. Specimens removed for examination at intervals of 4, 11 and 22 months showed evidences of renal infection in each case. Zeit,[10] in reviewing the morphologic findings from the experimental material of Martin and of Peterson, who also transplanted dog's ureters into the intestinal tract, observed that infection of the kidneys eventually took place. Sweet and Stewart [11] concluded from their researches on animals that the bacteria traveled upward via the lymph channels, after having demonstrated abundant lymphatic anastamoses in the mucosa, submucosa and external coats of the bladder and ureters, and the entire renal structures. They felt that they could exclude the blood vessels as a possible avenue of infection because: (1) these, for the most part, emptied into the general venous circulation; (2) the infection could never be traced along the mucosa, but was found in the lymph channels; (3) if the continuity of the lymphatics was interrupted, the infection did not reach the kidney at all; (4) if the ureteral lumen was closed to infection but the lymphatics left undisturbed, the infection ascended in the usual manner; (5) finally, if the renal pelvis was connected directly with the intestine, no infection of kidney occurred. Eisendrath and Kahn,[12] after injecting bacteria into unobstructed, nontraumatized bladders of rabbits and dogs, felt that the organisms ascended along the periureteral lymphatics to the kidney pelvis. Helmholz and Beeler,[13] after several series of inoculation experiments in rabbits, were of the opinion that the infection could travel both by way of the lumen of the ureter and by its lymphatics. David,[14] from the results of his work on dogs, felt that it was possible to infect the upper urinary tract in an unobstructed bladder, the micro-organisms gaining the renal tissues by direct extension through the ureteral lumen.

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9. Martin: Implantation of Ureters Into Rectum, J. A. M. A. **32**:159 (Jan. 28) 1899; **32**:237 (Feb. 4) 1899; **32**:709 (April 1) 1899.

10. Zeit: Pathology and Bacteriology of Ureteral Anastomosis, Tr. Chicago Path. Soc. **4**:210, 1899-1901.

11. Sweet and Stewart: The Ascending Infection of the Kidneys, Surg., Gynec. & Obst. **18**:460 (April) 1914.

12. Eisendrath, D. N., and Kahn, J. V.: Rôle of the Lymphatics in Ascending Renal Infection, J. A. M. A. **66**:561 (Feb. 19) 1916.

13. Helmholz and Beeler: Ascending Infection of the Urinary Tract, Tr. Am. Pediat. Soc. **30**:196 (May) 1918; Experimental Pyelitis in the Rabbit, J. Urology **2**:395 (Oct.) 1918.

14. David: Ascending Urinary Infections, Surg., Gynec. & Obst. **26**:159 (Feb.) 1918.

Consequently, by whatever avenues the infection is accomplished, it is generally recognized that the ascending type of renal infection is an entity. This would seem to be clearly indicated in the case herein reported, in which the sequence of pathologic changes evidently was ureteritis, pyelitis, pyelonephritis, pyonephrosis and perinephric abscess, with and without calculus formation. However, inasmuch as there was plenty of damage in all coats of the ureteral wall and the renal pelvis, and a marked inflammatory involvement of the perivascular lymphatics in these structures, one would be at a loss to ascribe the pathway of infection to a single anatomical structure.

The case is unique in many ways, and when it is viewed in its entirety, the wonder is not that these patients die of ascending renal infection, but that some are able to withstand it for so long a time. With the finding of such a ready communication between rectal cavity and ureteral orifices, the case, too, suggests that the time-honored altercation as to the best method of rectal implantation of the ureters with a view to preserving sphinteric control is of more academic than practical importance, and that, in exstrophy of bladder, the surgeon finds himself between Scylla and Charybdis, where, of the evils presented, he must choose the least.

CONGENITAL ABNORMALITIES OF THE INTESTINE

INTESTINAL APLASIA: ATRESIA OF THE RECTUM AND ANUS COMPLICATED BY RECTOVESICAL FISTULA, WITH A REPORT OF CASES *

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MINNEAPOLIS

INTESTINAL APLASIA

The rarity of intestinal occlusion is shown by the reports from St. Petersburg (Petrograd, Leningrad) where only nine cases were found in 150,000 necropsies, from Vienna where two cases were found in 110,000 necropsies, from Moscow where thirty-six cases in 65,000 necropsies and from Copenhagen where two cases in 41,000 necropsies were found. The usual ratio is considered 1 : 20,000, although it is well known that many infants are not examined post mortem, but are said to have died of a "weakened condition." The foregoing reports include occlusion from all sources. Those cases in which the condition is due to aplasia would be even more uncommon. It is important, therefore, that more postmortems should be sought, and it is with this plea in mind and because of the rarity of occurrence of intestinal aplasia that this paper is submitted.

No attempt will be made to discuss all of the various types of congenital intestinal obstruction due, for example, to contractions, Meckel's diverticulum, hernia, volvulus, inspissated meconium, infected mucus plugs, tumors, inflammation, or intussusception.

To avoid confusion it may be well to give definitions of the terms often used in referring to abnormalities of the hollow viscera. The term atresia refers to the imperforation of an opening or of a canal; aplasia is defective development in a tissue; stenosis refers to a narrowing or constriction, and occlusion is the blocking of an opening.

In order to understand better the abnormality mentioned it may also be well to review briefly the normal development of the intestine.

Embryology.—According to Prentiss,[1] the spherical vitelline sac forms blind entodermal tubes, namely, the foregut and hindgut. The midgut between these opens into the yolk sac, the stalk of which is lost at 7.5 mm. The ends of the foregut and hindgut touch ectoderm to form the pharyngeal membrane and the cloacal membrane, respectively. The foregut later differentiates into the pharynx, esophagus, respiratory

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1. Prentiss, C. W.: Normal Embryology, W. B. Saunders Company, 1915.

organs, stomach, duodenum, jejunum and part of the ileum. The hindgut extends to the cloaca, which subdivides into the rectum and urogenital sinus, into which the allantois opens (2 mm.) and finally from this portion (the hindgut) are developed the ileum, cecum, colon and rectum (Fig. 1). Omitting the structures not considered in this paper, the intestine in a 5 mm. embryo consists of the duodenum and the cephalic and caudal limbs of the intestinal loop, the ventral bend of which connects with the yolk stalk and then continues caudally to the cloaca. The persistent yolk stalk marks the site of a pouch, Meckel's diverticulum, often a cause of intestinal occlusion in infants. At from 5 to 9 mm., a bulging in the caudal limb of the primary intestinal loop marks the "anlage" of the cecum, the boundary between the large and small bowel. This later becomes the cecum and the vermiform process (Fig. 2). Then follows torsion and coiling due to elongation, and six primary intestinal loops form, which are present in the umbilical cord at 17 mm. At 42 mm., the intestine has returned to the abdominal cavity through a small aperture which closes soon after. At from 10 to 30 mm., vacuoles appear in the wall of the duodenum, and epithelial septums close the canal. The remainder of the canal remains open, although vacuoles appear in the epithelium. Villi first appear in the jejunum at 130 mm. and are found throughout the small intestine. Intestinal glands first appear in the jejunum at 91 mm. and in the duodenum at 130 mm. Circular muscle layers form at 10 to 12 mm., and the longitudinal fibers are not distinct until 75 mm. Prentiss mentions that the impervious duodenum of the embryo may persist as a congenital anomaly.

Because of the importance ascribed to the mesentery in cases of intestinal aplasia, the normal development of this will also be briefly reviewed from the same author.

During development, when the headfold and foregut of the embryo first present, the layers of the splanchnic mesoderm containing the heart tubes are folded together ventral to the foregut and form the ventral mesentery. Caudal to this grows the liver, and from this part of the mesentery develop the lesser omentum dorsally and the falciform ligament anteriorly. There is no ventral mesentery caudal to the liver because of the presence of a yolk sac early in the development. Dorsal to the gut, the splanchnic mesoderm folds over on each side to form the complete dorsal mesentery. It is this which particularly interests us (Fig. 2). Primarily, this extends in a median sagittal plane. The mesentery of the intestine is carried into the umbilical cord between the loops previously mentioned. During the elongation and looping of the intestine, the cecal end of the large intestine lies cranially and to the left with the small intestine caudally and to the right (Fig. 2); but on the return of the intestinal loop into the abdomen from the umbilical cord, the cecal end of the colon lies to the right and the transverse colon crosses the duodenum. The primary loops of small intestine lie caudal and to

the left of the ascending colon. Thus, the mesentery has undergone torsion about the base of the mesenteric artery as its axis, and it spreads out like a fan. Prentiss adds that malformations of the mesentery may be due to the persistence of simpler embryonic conditions usually correlated with the defective development of the intestinal canal.

Etiology.—Various theories regarding the etiology of intestinal atresia have been advanced. Some of the causes given are: (1) the presence of adhesive bands; (2) the blocking of mesenteric vessels, as

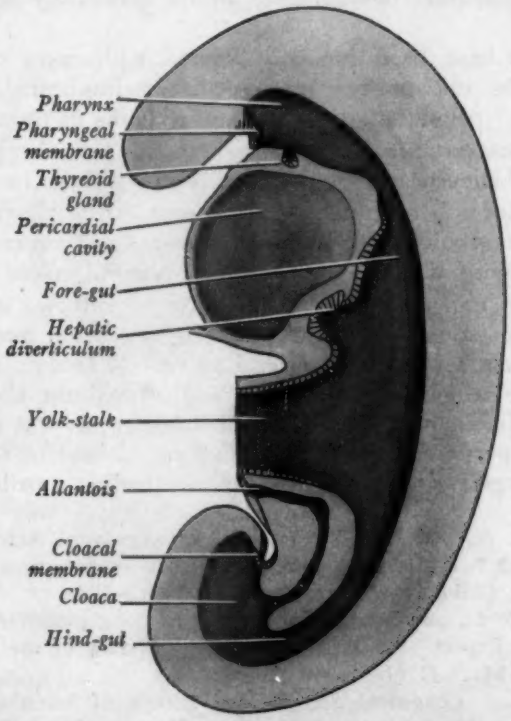

Fig. 1.—Diagram in median sagittal section of the alimentary canal, pharyngeal and cloacal membranes of a 2.5 embryo, after Thompson (from Prentiss).

in sclerosis; (3) infections, such as syphilis or nonspecific infections; (4) volvulus; (5) pressure of surrounding organs, and (6) embryologic defects.

Kahn [2] mentions atresia brought about by pressure of amniotic bands, but, in his own case, he favors the theory of epithelial occlusion as the cause. Landau [3] also mentions a case of bands of adhesions

2. Kahn, M.: Report of a Case of Atresia of the Ileum with Microcolon, J. A. M. A. **76**:721 (March 12) 1921.

3. Landau, H.: Kongenitale Darmverschlulles Infolge von Atresie des Ileums, Deutsch. med. Wchnschr. **56**:1195, 1920.

leading from blind ends. Owen's [4] case showed a band of mesentery blending with the urachus and a remnant of the vitteline artery.

Blocking of the mesenteric vessels has its advocates in Davis and Poynter,[5] who found the vessels to be sclerosed and accompanied by secondary degeneration and dissociation of the mucosa. Sironi [6] quotes Wiss, who reported the absence of the afferent mesenteric artery. Keith [7] believes that the V-shaped gap often found in the mesentery may be caused by volvulus with the wide separation of the blind ends which follows. Quinlan [8] found no evidence of arterial thrombosis. Our own case revealed normal blood vessels in the mesentery surrounding the defect.

Infections have been found associated with cases of atresia, and syphilis is the one most often specifically implicated. Rendu and Wertheimer's [9] patient gave a history of syphilis in the mother, but, in their case, nonspecific infection in the intestine was also present. Likewise, Holt [10] believes that syphilis may be an etiologic factor.

Nonspecific infection may play a part in producing mechanical obstruction, as shown by Hughes,[11] whose case presented a blocked transverse colon with a perforating stercoral ulcer three-fourths inch (1.27 cm.) in diameter. In the case of Bullowa and Brennan,[12] inspissated meconium was found blocking the ileum one and one-half inches (3.81 cm.) from the ileocecal valve. Theremin, in 1877, made an exhaustive review of the subject and offered the theory that congenital stenosis might be caused by inflammation, as was shown in cases of enteritis, peritonitis and strangulation. Ichenhauser [13] reports a case with gangrenous hernia of a blindly ending small gut near the

4. Owen, S. A., and Lake, N.: Report of a Case of Antenatal Intestinal Obstruction with Some Remarks on Other Forms of Intestinal Obstruction in Infants, Brit. J. Child. Dis. **17**:115-126 (July-Sept.) 1920.

5. Davis, D. L., and Poynter, C. M. W.: Congenital Occlusions of the Intestines with Report of a Case of Multiple Atresia of the Jejunum, Surg. Gynec. & Obst. **34**:35-41 (Jan.) 1922.

6. Sironi, L.: Congenital Stenosis and Atresia of Intestine, Riv. di Clin. Pediat., Florence **20**:65 (Feb.) 1922; abstr. National M. & S. Survey **9**:697 (May) 1922.

7. Keith, A.: V-Shaped Gape in Mesentery, Brit. M. J. **1**:301, 1910.

8. Quinlan, W. S.: Congenital Malformations of the Intestine, Atresia and Imperfect Anus: Report of Twenty-Seven Cases, Boston M. & S. J. **87**:870-875 (Dec. 14) 1922.

9. Rendu, A., and Wertheimer, P.: Case of Congenital Obstruction of Small Intestine, Arch. de méd. d. enf. **25**:349 (June) 1922; abstr. Int. Med. Survey **4**:147 (July) 1922.

10. Holt, L. E.: Diseases of Infancy and Childhood, New York, D. Appleton & Co., 1917.

11. Hughes, E. E.: Acute Intestinal Obstruction Due to Inspissated Meconium, Brit. J. Child. Dis. **19**:32-33 (Jan.-March) 1922.

12. Bullowa, J. G. M., and Brennen, R. E.: Intra-Uterine Intestinal Obstruction from Inspissated and Impacted Meconium, J. A. M. A. **73**:1882 (Dec. 20) 1919.

ileocecal valve. Rendu and Wertheimer's case showed an obstruction near the cecum connected with the atrophied colon by a cord, the intestinal structure of which was lacking. It could not be dilated, according to this author, because of the *Bacillus smegma* present (in our cases there was no such cord).

Volvulus and the pressure of surrounding organs are mentioned. Leischner's [13] case showed 180 degrees of torsion and such histologic findings as hyalinization, calcareous deposits and necrosis much older than the baby's age (5 days), so that she considers this a case of intrauterine stenosis, which may even have existed before volvulus took place.

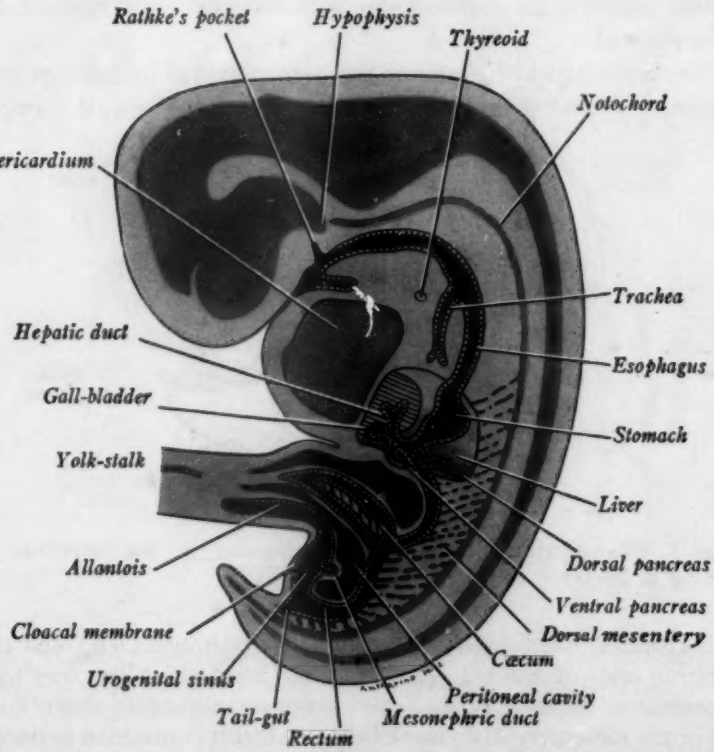

Fig. 2.—Diagram, in median sagittal section, of the digestive canal of a 9 mm. embryo, adapted from Mall (from Prentiss).

Of all the theories proposed, the developmental appears the most logical. For, as it has been noted in the normal embryology, there is one stage in which at least the duodenum is completely occluded by proliferating epithelium to form septums and compartments. This is more pronounced in lower animals. The persistence of one or more of these would explain the septum type of obstruction reported by

13. Leischner, Frieda: Ueber Kongenitalen Stenose und Achsendrehung des Dunndarmes, Beitr. z. path. Anat. u. z. allg. Path. **67**:28-39, 1920.

Morley,[14] Schroder [15] and others. Then, as Forssner has suggested, if one considers the septum a "thin bridge" and a cord connecting two parts of the intestine a "thick bridge," the embryologic theory seems quite plausible. The cases reported by Quinlan, Kahn, Rendu, Wertheimer and Sironi would fall into this group. A third group, in which the blind ends of the gut are free is mentioned, and cases reported by Keith, Vander Bogert,[16] Landau, Davis and Poynter and ourselves fall into this class. This type of case, because of the V-gap in the mesentery, usually brings the mesentery and its blood supply into discussion as a causative factor, but it should probably be considered as Prentiss suggests, as correlational with defective development of the intestinal canal.

The classification of Forssner has been amplified by Spriggs (quoted by Bryan [17]) who gives the following types: (*a*) simple narrowing;

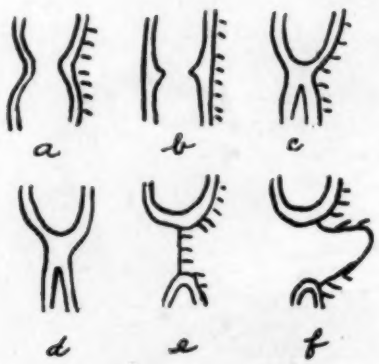

Fig. 3.—Classification of defective development of the intestinal canal, according to Forssner (after Spriggs).

(*b*) perforated diaphragm; (*c*) complete diaphragm; (*d*) short band connecting ends of bowel; (*e*) thread-like band along the free edge of the mesentery, the ends of the bowel being some distance apart, and (*f*) a gap in the mesentery also, there being no direct connection between the ends of the bowel (Fig. 3).

The fact that septums and epithelial occlusions form most abundantly in the duodenum might account for the statement that most of the

14. Morley, J.: Congenital Occlusion of Ileum, Brit. J. Surg. **9**:103-110 (July) 1921.

15. Schroder, C. H.: Congenital Obstruction of Duodenum: Report of a Case, J. A. M. A. **78**:1039-1041 (April 8) 1922.

16. Vander Bogert, F.: A Case of Congenital Atresia of Small Intestines, Arch. Pediat. **35**:45 (Jan.) 1918.

17. Bryan, R. C.: Congenital Occlusion of Small Intestine, Am. J. Surg. **37**:297 (Dec.) 1923.

obstructions occur here, according to the report of Salzmann,[18] which is supported by Quinlan, who encountered four of the duodenum to two of the ileum. Sironi, Schroder and Iversen[19] each report one in the duodenum. Others, however, report that they are most frequent in the ileocecal region. This seems to be borne out by a majority of the writers. Ichenhauser[20] quotes Zahl, who studied two hundred cases of aplasia and found most of them near the ileocecal valve. A few other writers, each reporting the finding of the anomaly in the ileum once, are Quinlan,[8] Kahn,[2] Rendu and Wertheimer,[9] Morley,[14] Vander Bogert,[16]

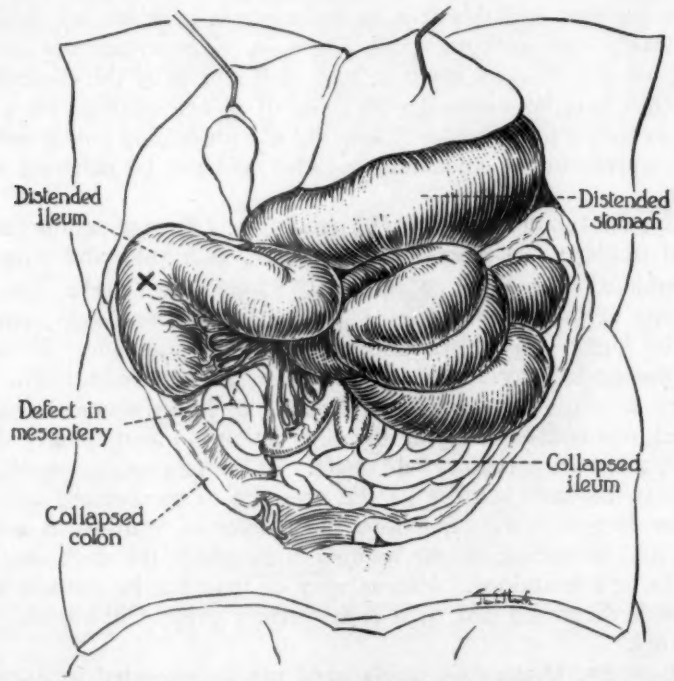

Fig. 4 (Case 1).—Appearance of the distended stomach and proximal ileum, the collapsed colon and distal ileum, and the defect in the mesentery in intestinal aplasia; *X* marks the point at which the ileostomy was made.

and Landau.[3] Sironi reports two of the jejunum, and Davis and Poynter one of the duodenojejunal region, and from their study of 392 cases they estimate that 15 per cent. of the cases show multiple defects. One of Davis and Poynter's cases showed five points of atresia.

18. Salzmann, Matilda: Ueber kongenitale duodenal Atresia, Monatschr. f. Kinderh. **19**:468-470 (March) 1921.

19. Iversen, Dahl E.: Ileus as a Consequence of Certain Congenital Anomalies, Hospitalstidende, Copenhagen, **65**:189 (April 25) 1922; abstr. J. A. M. A. **79**:254 (July 15) 1922.

20. Ichenhauser, M.: Ein Fall von Atresie des Dermes mit Varfall einer gangranosen Darmschlinge durch einen Spalt in der Bauchwand beim Neugeboren, Deutsch. Ztschr. f. Chir. **163**:417 (June) 1921.

Landau [3] places the jejuno-ileal junction first in frequency, the duodenum next and the large intestine, except for defects of the rectum and anus, last. Quinlan [8] believes that a ratio of one to four exists between intestinal atresia and imperforate anus. If the statistics showing most defects in the ileum prevail, it would seem that septum formation, at least, does not predispose to the aplasia. The greater frequency of anal anomalies can, of course, be explained by the failure of the anal membrane to disappear normally. This will be discussed later.

Symptoms and Diagnosis of Aplasia.—Having considered the embryology and admitting that the etiology of intestinal aplasia is unknown but that probably it is an embryologic accident, we shall next discuss briefly the symptoms and diagnosis. The symptoms naturally are those of intestinal obstruction, and such is usually the clinical diagnosis. They may be identical with those of pyloric obstruction without a tumor as shown by Schroder [21] who did a jejunostomy and at necropsy found a septum in the duodenum which could not be palpated during operation.

Briefly, the symptoms are: (1) vomiting; (2) obstipation (at least abnormal stools); (3) abdominal distention, as a rule, and with it, at times, umbilical distention; (4) anuria; (5) collapse (late). The onset of vomiting varies with the site of the obstruction, as a rule appearing earlier the higher the obstruction occurs in the intestine. It usually appears the second day. In our case, it was present from birth. Bile, blood, mucus, meconium or feces may be found in the emesis. If bile is vomited, one is safe in saying the obstruction is infrapapillary unless, of course, there are accessory bile ducts. In such a case, the stools may, nevertheless, be dark because of the presence of meconium, according to Vander Bogert,[16] who mentions the danger of waiting in such an instance, and according to our own case, in which the dark stool was thought to be bile-stained. Icterus may or may not be present and is of very little diagnostic aid, as it is frequently present in normal newly born infants.

The complete absence of stools need not be expected in a case of intestinal aplasia, as our own case will illustrate; but a small stool made up mainly of mucus and meconium may be passed. Lanugo, however, are not found, according to Bryan [17] and Walz, who is quoted by Salzmann.[18] Thus, stools should be tested for bile and lanugo and, if these are not found, obstruction is very likely to be present. Lanugo appear at about the fifth fetal month, and their absence gives some idea as to duration of the obstruction. Sterile meconium also points to a complete atresia.

Abdominal distention may be absent if the obstruction is high, and there may really be, in such a case, a retraction of the abdomen, according to Schwalbe, who is quoted by A. V. St. George.[22]

21. Schroder, C. H.: The Diagnosis of Congenital Obstruction of Duodenum, J. A. M. A. **82**:687 (March 1) 1924.

22. St. George, A. V.: Congenital Intestinal Obstruction with Report of a Case, Am. J. Dis. Child. **15**:354 (May) 1918.

Umbilical protrusion may accompany the distention. The onset of distention may be noted the first day, and rigidity of the abdomen was noted by Salzmann in the lower left quadrant rather than over the epigastrium.

Visible peristalsis of the intestine was noted by Vander Bogert and was also recorded in our case.

In addition to vomiting, Peterson [23] mentions a fall of blood pressure, temperature disturbance, impaired renal function, blood changes, such as delayed coagulation and a high nonprotein nitrogen, and collapse in describing the picture of obstruction. Similar blood findings have also been shown to exist in adults by Connors, Killian and Eisberg.[24]

Diagnosis.—The diagnosis of intestinal obstruction may be obvious from the foregoing symptoms, but the site of obstruction is most often established only at operation or necropsy. In considering the differential

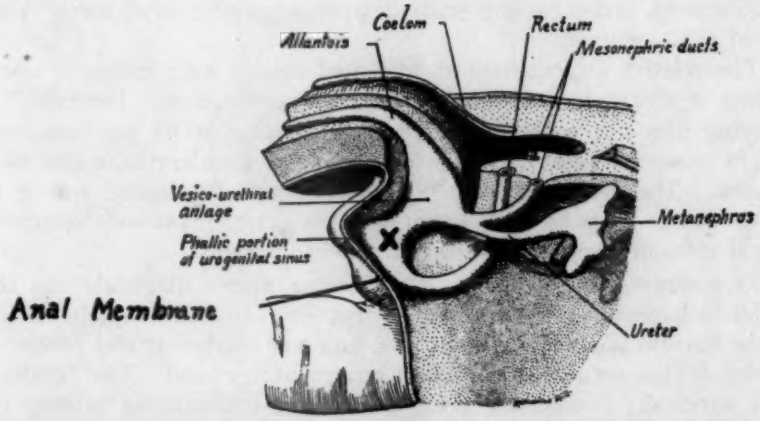

Fig. 5.—Reconstruction of the caudal portion of an 11.5 mm. embryo, showing the differentiation of the rectum, bladder and urethra, after Keibel (from Prentiss).

diagnosis, Bullowa and Brennen [12] bring out the point that if a proctoscopic examination with a cystoscope shows rhythmic discharge from the bowel, then the musculature is continuous and the obstruction may be a plug of mucus or meconium such as existed in their case, and a break in continuity of the bowel does not exist. Bryan [17] states that rectal atresia always occurs within 2 inches (5.08 cm.) of the anus and thus should offer no difficulty in the differential diagnosis. Schroder,[21] in a recent paper, adds a point in differentiating duodenal from pyloric

23. Peterson, E. W.: Acute Intestinal Obstruction in Infancy and Childhood: A Brief Review of Fifty-Five Cases, Surg. Gynec. & Obst. **35**:436-439 (Oct.) 1922; abstr. Int. M. & S. Survey, 1922.

24. Connors, J. F.; Killian, J. A., and Eisberg, H. B.: Chemical Changes in Blood in Intestinal Obstruction, Proc. Soc. Exper. Biol. & Med. **20**:357 (April) 1923.

obstruction by inserting a small catheter and washing the stomach until clear fluid returns and then pushing the catheter through the pylorus, thus determining its patency. If after this a gush of bile-stained fluid follows, one is most likely dealing with a duodenal obstruction.

Very few radiologic reports have been published, and Peterson [23] makes a plea for a more general use of this procedure, which we think quite apropos. The signs to be looked for are dilatation and filling proximal to the obstruction, with prolonged retention and ineffectual peristalsis. Shroder calls attention to the fact that, as in the case of the adult, the duodenum is best shown by having the child lie on its abdomen or by having him stand. The following point has occurred to us. Might it not be wise to advocate the routine administration of small amounts of barium sulphate at birth and, if symptoms of obstruction present, gain some information from the roentgen ray within twenty-four hours? Up to the present, such cases have remained too long unrecognized, and a barium study delayed may, of course, merely mean loss of valuable time.

The relative infrequency of intestinal aplasia may influence one in making a diagnosis between this and intussusception. Peterson,[23] in studying fifty-five cases of obstruction, found that 83 per cent. were due to intussusception, which was twice as prevalent in males as in females. The remaining small percentage was distributed among the various other types of obstruction, and the rarity previously mentioned should influence one in making a definite diagnosis.

Treatment and Prognosis.—Considering that a diagnosis has been established, usually of intestinal obstruction (unless definitely decided by the barium study), our next problems are treatment and prognosis. Obviously, the results of expectant treatment are fatal. The results of cases surgically treated are but slightly more encouraging as only two patients thus treated, those of Fochen and Ernst, have recovered, according to Bryan [17] and Sironi [6] in a study of cases reported in the literature. Therefore, a bad prognosis must be given.

Duodeno-enterostomy, when indicated, is the procedure advocated, and ileostomy or jejunostomy have not been credited with saving any of the babies. The question of anesthesia is one considered of importance by many of the writers. Bullowa and Brennen used chloroform, Waugh [25] used spinal anesthesia, and Landau, Clogg and Bryan advocate the use of local rather than ether. Procain was used in our case, and the baby withstood the operation satisfactorily; our mistake consisted in postponing surgical treatment too long.

REPORT OF CASES

History.—J. C., a boy, aged 2 days, was brought to St. Mary's Hospital, Jan. 17, 1920. In consultation with Dr. C. A. Lapierre, I first saw the child when it was 54 hours old. Since birth there had been no bowel movement. Vomiting began a few hours after delivery and was continuous thereafter,

25. Waugh: Lancet **1**:427, 1912.

consisting of black material (meconium). He nursed a few times the first day, but refused to do so after the second day. A rectal tube was passed 5 cm. by the family physician, Dr. Lapierre, but no meconium was expelled.

Examination.—Examination showed a fairly well nourished child with a moderately distended and tympanitic abdomen. No masses were palpable, and the liver edge could be felt about 2 cm. below the costal margin. Up to this time, there had been no discharge whatever from the anus, but during the examination a plug of gelatinous material, dark and light yellowish brown and the size of a hazel nut, was expelled (2.5 cm. by 1 cm.). It was thought that this was the cause of the obstruction, and operation was deferred. The child was removed from the hospital by its parents. On Jan. 21, 1920, he was returned. The baby's abdomen was more distended, and vomiting was more persistent. There had been no further bowel movements, and active peristalsis was visible. He was cyanotic and in poor condition. The diagnosis of intestinal obstruction was considered certain at this time.

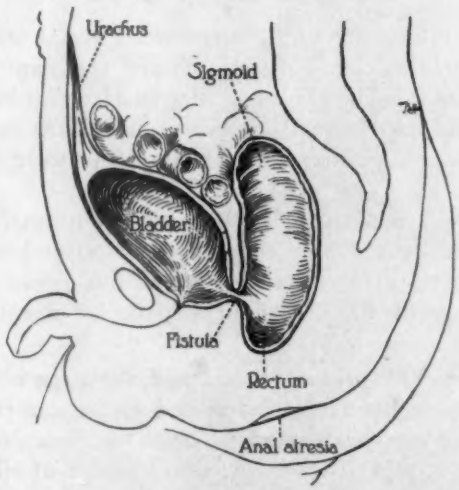

Fig. 6 (Case 2).—A sagittal view of the rectum and anus complicated by rectovesical fistula, showing the condition existing at birth.

Operation.—Enterostomy was performed, with local infiltration, using 15 c.c. of a 0.7 per cent. solution of procain-epinephrin. A right-sided gridiron incision was made, and the first presenting loop of distended bowel was brought out and opened. A rectal tube was sutured into it. The wound was partially closed. Much gas and fecal material was discharged during the following night, but the baby gradually grew weaker and died the next day, twenty-one hours after operation and four days after birth.

Necropsy.—Examination by the University of Minnesota Department of Pathology, showed several loops of distended small bowel lying directly beneath the parietal peritoneum (Fig. 4). The stomach, duodenum and jejunum were dilated. At a point about 75 cm. from the stomach, the dilated small bowel came to a blind ending. It will be noticed that this is not the most common site of obstruction, according to the previous findings of Tandler, Leichtenstern, Salzmann and Quinlan, who located it most commonly in the

duodenum. The bowel wall presented an incision 3 cm. proximal to the blind ending representing the enterostomy opening (Fig. 4, *X*). The continuity of the bowel was completely absent at this point, the blind end of the distal portion being found 5 cm. posterior and to the left. Some cases, Rendu and Wertheimer's, may show a cord connecting the blind ends (Fig. 3, *E*). A break in continuity of the mesentery was also present, giving a V-shaped defect. The greater distal portion of the small and large bowel were collapsed and apparently much smaller than normal, showing a twisted, pale pink tube the size of a lead pencil but containing a lumen throughout. The cecum and entire colon were in a normal position. There were no other gross abnormalities.

Microscopic sections taken from the free edge of the mesentery, which connected with the proximal blind ends, showed an entire absence of any intestinal tissue. The blood vessels showed no changes, and a lymph gland in this region was found to be normal.

ATRESIA OF THE ANUS AND RECTUM COMPLICATED BY RECTOVESICAL FISTULA

McDonald,[26] estimating the occurrence of rectal atresia, states that it occurs once in 5,000 new-born infants and Quinlan [8] gives its occurrence once in 15,000. The Boston Lying-In Hospital had one case, and the Chicago Lying-In Hospital two cases in 10,000 new-born infants. Of twenty-eight cases Quinlan found twenty-four were in boys and four in girls.

Such a close relationship exists between the normal development of the anus and the urinary tract that, in order to understand better the combined anomalies (atresia and fistula) of this region, a brief review of the embryology of these structures given by Prentiss [1] may prove helpful.

Embryology.—As previously mentioned, the large intestine continues as a tube from the cecum to the cloaca, and the various parts of the large intestine develop from the former, whereas the cloaca or caudal expansion of the hindgut takes part in the development of the allantoic stalk (urachus) ventrally extending from the cloaca to the umbilicus (Fig. 2). The mesonephritic ducts, buds of which later represent the ureteric anlage of metanephroi, develop laterally from the cloaca at 5 mm. The cloacal membrane consisting of entoderm and ectoderm is formed as the cloaca (entoderm) comes in contact ventrally with the ectoderm. Then a saddle-like partition forms between the intestine and the allantois which grows caudally, dividing the cloaca into a dorsal rectum and ventral urogenital sinus (10 to 15 mm.). As this partition fuses with the cloacal membrane, it divides it into an anal and a urogenital membrane (Fig. 5). The urogenital sinus differentiates into a vesico-urethral anlage, which receives the allantois and the mesonephric ducts and later forms the bladder and part of the urethra, and the phallic portion, which later forms the greater portion of the urethra.

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26. McDonald, A. L.: Imperforate Anus, with Report of a Case, Am. J. Surg. **36**:115 (May) 1922; abstr. Internat. M. & S. Survey **3**:1172 (June) 1922.

Thus, it can readily be seen how failure of perforation may take place at the anal membrane, and the possibility of a persistent connection between the bowel and the genito-urinary tract is also explained (Fig. 5, *X*). Cunningham makes the point that there may be a deficient hind-gut with a considerable interval between the proctoderm and the gut (defectus recti partialis or totalis).

Etiology.—Why there should be this arrest in development is unknown, and heredity is said not to be a factor. Syphilis again is

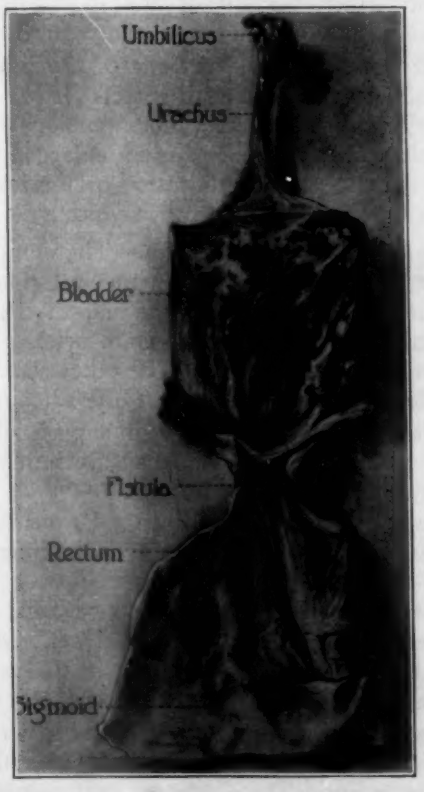

Fig. 7 (Case 2).—Specimen obtained at necropsy. The bladder, fistula, rectum and sigmoid are open.

mentioned by some as playing an etiologic part. At least, syphilis was present in Lantuejoul's and Walter's [27] and Cristol's [28] cases.

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27. Lantuejoul and Walter, H.: Anal Malformation in a Case of Congenital Syphilis, Bull. et mém. Soc. anat. de Par. **92**:54 (Feb.) 1922; abstr. Internat. M. & S. Survey, June, 1922.

28. Cristol, V.: Abnormal Vesical Urination of the Rectum in Infants, J. d'urol. med. et chir. **13**:243 (April) 1922; abstr. Internat. M. & S. Survey **4**:110 (July) 1922.

Associated with atresia of the anus are usually other anomalies such as a persistent cloacal duct or, as Quinlan found, congenital absence of the kidney and intestinal atresia. There may be a markedly prolonged sinus extending within the raphe beyond the anus to the scrotum, according to the reports of McDonald [26] and Vanverts.[29] A case simliar to this was recently shown by Dr. Frank Mach at St. Mary's Hospital, Minneapolis. In some instances, these sinuses have been known to extend to the scapular region (Dinsmore) or to the neck, even opening in the face (Bils) as mentioned by Quinlan.[8]

Depending on which portion of the saddle-like partition of the cloaca is defective, with a resulting fistula between the rectum and genito-urinary tract, there may develop any one of the following fistulas with or without atresia of the anus: (1) rectovesical fistula; (2) recto-urethral fistula (prostatic or membranous portion) or (3) rectovaginal fistula. In addition, such anomalies as an enlarged cloaca, reported by Cruickshank,[30] or a congenital duplication of the lower segment of the digestive tract as reported by Glatzel [31] may occur.

Symptoms and Diagnosis of Atresia.—The symptoms of atresia of the anus without a fistula leading from the intestinal tract differ from those of the same condition with a fistula, in that the latter shows meconium or fecal discharge from the genito-urinary tract, and the toxic symptoms may be delayed or absent. The other common symptoms are: (1) vomiting (late); (2) distention of the abdomen with tympany; (3) active peristalsis, and (4) engorged superficial veins (Quinlan). One case in which there was pronounced abdominal distention caused by fetal ascites is reported by Cruickshank.

The diagnosis should not be difficult and a routine attempt to take a rectal temperature at birth would result in an immediate diagnosis as to nonpatency of the anus.

In case a fistula is present, meconium or fecal material may discharge from either the vagina or the urinary tract. In males, it naturally comes from the bladder and in females from the vagina, in which the fistula more often opens, according to Cristol.[28] Here, as Glatzel [31] has shown, there may be a duplication of the lower intestinal tract so that one limb of the fistula opens into the vagina and the other leads to the anus, which is patent. The discharge may persist for an indefinite time, and Glatzel's case was not treated surgically until the patient had reached the age of 12 years.

The site of the opening into the urinary tract may be quite accurately located if the relation of the meconium or fecal discharge to urination

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29. Vanverts, J.: Imperforation of Anus: Prolongation of Intestine to a Point Near the Scrotum Through the Subcutaneous Canal, Bull. Soc. d'obst. et de gynéc. de Paris **10**:396 (No. 6) 1921; abstr. Internat. M. & S. Survey, 1921.

30. Cruickshank, J. N.: Persistent Cloaca with Imperforate Anus as a Cause of Fetal Ascites, Brit. M. J. **2**:980 (Dec. 10) 1921.

31. Glatzel: Congenital Duplication of Lower Segment of Digestive Tract, Polska Gaz. lek. Cracow **1**:667 (Aug. 20) 1922.

is noted. If the bowel content discharges irrespective of urination, the fistula opens into the urethra and not the bladder, as mentioned by McCown.[32] If it occurs primarily during urination, one can conclude that it is washed out of the bladder. Vomiting usually suggests some other intestinal defect with obstruction higher up; the diagnosis should be made before this occurs, in cases of simple atresia of the anus.

Treatment of Atresia of the Anus and Rectum.—This depends on the extent of the anomaly and the presence of other anomalies, such as atresia of some other part of the intestine, or the presence of a fistula communicating with the urogenital tract. The simplest cases merely require puncture of the anal membrane such as was done with scissors by McDonald.[26] Nine months later, however, he found obstructive symptoms, and at a second operation he dilated the anus In Wathen's [33]

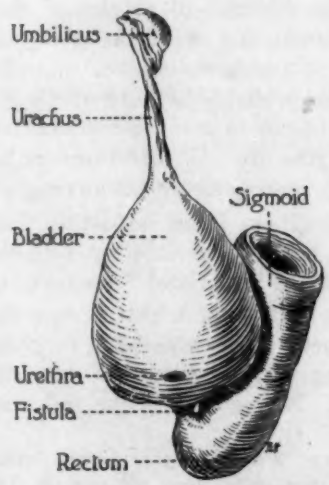

Fig. 8 (Case 2).—Surface view of the specimen obtained at necropsy (schematic).

case which was complicated with a rectovaginal fistula, a sound was merely passed into the rectum through the fistula and the anal membrane ruptured with a good result.

When atresia of the rectum in combination with an imperforate anus is present, it is necessary to unite the anal opening with the blind end of the rectum higher up in the pelvis, after bringing it down. If there is a congenital absence of the rectum, as in McCown's [32] patient, a higher segment of the bowel may be brought down to make an artificial

32. McCown, O. S.: Congenital Absence of the Rectum with a Communicating Sinus Between the Bowel and the Urinary Tract, J. Tennessee M. A. **15**: 262 (Oct.) 1922; abstr. Internat. M. & S. Survey **4**:970 (Sec. 2, Dec.) 1922.

33. Wathen, J. R.: Imperforate Anus. Case Report, Kentucky M. J. **19**:827 (Dec.) 1921; abstr. Internat. M. & S. Survey **3**:90 (Jan.) 1922.

anus. In his case, it was also necessary to close a sinus to the urinary tract. This was done by the perineal extraperitoneal route.

The perineal operation, done originally by Bell in 1787, should be tried first, as it is the most simple. In many cases, the blind end of the rectum is visible and can be brought down with ease and with a good result. In Oppel's [34] case he sutured the posterior half of the rectum to the anus and packed the anterior half away. At times, however, the terminal bowel is inaccessible, and colostomy is advised by some. Bevan, however, believes that only in the case of recto-vesical fistula should this be done.

The abdominal method of Trendelenburg is advocated by Klumper [35] for rectovesical fistula, and an incision along the superior border of the symphysis is used, freeing the prevesical space, repairing the bladder and draining.

The prognosis in the case of atresia of the anus, with or without rectal fistula communicating with the genito-urinary tract, is not as grave as is atresia of the bowel higher up. The fatality is greater in males than in females probably because of the greater number of urinary fistulas, which predispose to a more frequent condition of stercoremia. Other frequent dangers are (1) infections, either urinary, peritoneal or pulmonary, and (2) postoperative hemorrhage.

The type of anesthesia is an important factor in more ways than one, and all types have been used by the surgeons reporting cases. Chloroform is advocated by Cristol [28] because it permits of light administration and allows straining, which is essential in locating the blind end. A local anesthetic is the anesthetic of choice with us, since it permits this same maneuver and yet offers less chance of pulmonary and other complications. It was used in the following case.

History.—A boy, aged 9 days, was first seen January 29, 1919. He was the ninth child and the other eight were all normal. Castor oil had been given every day by the parents to produce a bowel movement, but it was finally discovered that the anus was closed. The child had been delivered without forceps, although labor had been difficult. He was not weighed, but he was considered below normal at birth by the parents, who also thought he had lost weight since. Strangely enough, to the parents, nothing abnormal except the absence of stools was noticed until the seventh day. Then he became irritable and cried a good deal. He nursed well until the eighth day. The urine had been very dark, and the mother stated that she believed "his bowels moved through his penis." Abdominal distention with resistance to palpation was present. There was no anal opening.

Operation.—Immediate perineal operation was decided on, with the baby in the knee-chest position. The line of incision was infiltrated, using 30 c.c. of

34. Oppel, W.: Atresia of the Anus and Rectum, Arch. f. klin. Chir. **119**:845 (May 13) 1922; abstr. Internat. M. & S. Survey **4**:273 (August) 1922.

35. Klumper, P.: Treatment of Vesicogenital Fistulas, Nederlandsch Tijdschr. v. Geneesk **2**:674-678 (Aug. 12) 1922; abstr. Internat. M. & S. Survey **4**:203 (Oct.) 1922.

a 1 per cent. procain-epinephrin solution. This permitted a 7 cm. incision for the splitting of the muscles, with very little crying on the part of the patient. A sac-like viscus was encountered which was thought to be either bladder or rectum, and it was observed that when the child cried gas escaped through the urinary tract. The sac was opened and was found to contain about 30 c.c. of dirty fluid (meconium). The edge of the opening in the viscus was sutured to the skin, and drains were inserted anterior to the coccyx. At the end of twenty-four hours, distention still presented and a colostomy was done, using local infiltration again for a left gridiron incision. Opening the bowel resulted in the discharge of a large amount of fecal material. However, the child died two days later.

Necropsy.—At the postmortem examination a slight localized peritonitis in the region of the loop of colon at the site of the operative wound was observed. The rectum was found dilated and extended into the pelvis, where it communicated with the urinary bladder by a short fistulous canal (Fig. 6). This canal was of a caliber that would permit the passage of a uterine sound. Figure 7 is a drawing of the specimen after the division of the wall of the lower sigmoid, rectum, fistula and bladder. It also presents the urachus and the umbilicus. Figure 8 is a schematic drawing of the same organs, showing their approximate size and position. Necropsy revealed the fact that the rectum had been opened at the primary operation.

In this case, it is entirely possible that a preliminary colostomy might have resulted in saving the child.

FRACTURE OF THE SKULL: ANALYSIS OF ONE HUNDRED AND SEVENTY-ONE PROVED CASES

DIAGNOSIS AND TREATMENT OF ASSOCIATED BRAIN INJURY

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AND
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LOS ANGELES

Clinical methods now enable us to secure more accurate information than was formerly possible in cases in which fracture of the skull is suspected; but because of limitations in the roentgen-ray diagnosis, particularly in connection with fracture at the base of the skull, considerable confusion still exists. The problem of the fractured skull becomes more clearly defined if when such a group of cases is analyzed instances are excluded in which a fracture is not definitely proved to exist. The complexity caused by associated injuries to the brain must be borne in mind. It is not unlikely that the experience of other clinicians, obtained when they apply such restrictions, will be similar to our own in finding that the number of cases in which a skull fracture may be actually demonstrated is overrated. Care must be taken to exclude doubtful cases if the problem is to be simplified, even though in limiting the study to proved cases much material is thereby sacrificed.

During the period of forty-one months from Oct. 16, 1920, to March 29, 1924, there were 580 patients with head injury admitted to the neurosurgical service of the Los Angeles General Hospital. The tendency to misjudge such cases is illustrated by the fact that, in most instances, "skull fracture" or "possible skull fracture" was the diagnosis on the admission sheet, while, in the majority of cases, only minor head injuries existed. In others, when the clinician had good reason to believe that at least a basal fracture had occurred, its existence could not be demonstrated. Our analysis, it seemed, would be more valuable if it were limited to cases of proved fracture. Accepting this standard, the all important symptoms of brain injury would be present and we not only could study the character of the fracture itself, but we could also view each case from the standpoint of associated intracranial damage. Many cases of suspected basal fracture with definite clinical signs had to be eliminated because the fracture was not actually seen. In a small number of fatal cases not included here, the fracture would have been revealed if a postmortem examination had been made. Gunshot wounds of the head are not included because we felt that, aside from the skull fracture and associated brain injury, the presence of a foreign body placed these cases in a category by themselves.

The cases comprising this report, 171 in number, represent a little less than one third (29.53 per cent.) of the total admissions to the hospital. Fracture of the skull was actually proved by roentgen-ray examinations in 158 cases, by operation in eleven and by necropsy in two. Of the 171 fractures demonstrated, 145 (84.8 per cent.) were linear, and twenty-six (15.2 per cent.) were depressed. These will be considered separately.

Right linear fracture.....93, or 64.2 per cent.
Left linear fracture.....52, or 35.8 per cent.
100 per cent.

Right depressed fracture.....12, or 46.2 per cent.
Left depressed fracture.....14, or 53.8 per cent.
100 per cent.

The ratio of the linear and depressed fractures for the 171 cases is as follows:

Right linear fracture.....93, or 54.4 per cent.
Left linear fracture.....52, or 30.4 per cent.
Right depressed fracture.....12, or 7.0 per cent.
Left depressed fracture.....14, or 8.2 per cent.

Table 1 classifies the fractures according to their location and the cranial bones involved. One hundred and five fractures (61.4 per cent.) of both types occurred on the right side, and sixty-six (38.6 per cent.) on the left. We are at a loss to explain the predominance of right-sided linear fracture.

TABLE 1.—*Classification of Fractures*

	Linear	Depressed	Total
Frontal	26	10	36
Anterior parietal	2	3	5
Posterior parietal	11	7	18
Occipital	14	1	15
Temporal	7	3	10
Frontal and anterior parietal	8	0	8
Frontal and temporal	2	1	3
Anterior and posterior parietal	4	0	4
Anterior parietal and temporal	6	0	6
Posterior parietal and occipital	1	0	1
Posterior parietal and temporal *	46	0	46
Frontal and anterior and posterior parietal	7	0	7
Frontal anterior, parietal and temporal	2	1	3
Occipital and posterior parietal and temporal	1	0	1
Anterior posterior, parietal and temporal	2	0	2
Occipital and temporal	5	0	5
Frontal and parietal and occipital	1	0	1
Right	93	12	105
Left	52	14	66
Total	145	26	171

* For purposes of description, the parietal and temporal bones have been divided arbitrarily into the anterior and the posterior half. This has been done because of the far greater number of fractures shown to have occurred in the posterior half (31.7 per cent. of all linear fractures).

CONSIDERATION OF LINEAR FRACTURES

In view of the impression long ingrained, that linear fractures seek the thinner portions of the skull and avoid the buttresses, an analysis

of our roentgen-ray findings was enlightening. It soon became apparent that the fracture line traversed the thicker portions of the calvarium as often as the thinner, and not infrequently actually invaded the so-called buttresses at the base. No direction existed which could be called constant, although in two locations, the occipital and the posterior parietotemporal, the fracture lines followed a fairly uniform direction. The posterior parietal region is well known as one of the thicker regions of the calvarium; yet in 35.8 per cent. of all linear fractures the line was found in this region and coursed down through the posterior half of the temporal bone toward the external auditory meatus, frequently extending into the heavy petrous portion below. In spite of the fact that the anterior half of the temporal bone, and perhaps of the parietal as well, is thinner than the posterior, fracture lines here were found much less commonly (a ratio of forty-six, 88.5 per cent. in the former, to six, 15.5 per cent. in the latter). Moreover, with occipital fracture, the line of fracture not infrequently appeared in the roentgen-ray plates to course actually through the reinforced margins of the foramen magnum. As reference to the foregoing table and to the diagrams which follow will show, we find that fracture lines may involve practically any cranial bone (often more than one) and run in almost every direction. In the linear series, the fracture occurred most frequently in the following order: posterior parietotemporal, 46 (31.7 per cent.); frontal, 26 (17.9 per cent.), and occipital, 14 (9.7 per cent.). The fracture was confined to one cranial bone in sixty cases (41.7 per cent.). Actual contact tracings of linear fractures from roentgen-ray films have been reproduced in composite drawings, showing their occurrence in both the right and the left, frontal and occipital positions. While linear fractures were traced as accurately as possible, some allowance must be made with regard to both location and direction because of the individual variation in skulls. In eleven instances, fracture lines were traced diagramatically from the description of findings at necropsy; therefore, they may not be accurate. In one instance, the fracture, a right temporal, was drawn in, as seen at operation. There were five instances of suture separation, two lambdoidal and three coronal; these have not been incorporated in these tracings, although they have been considered to be in the category of linear fractures.

DEPRESSED FRACTURES

Of the twenty-six depressed fractures, twelve (46.2 per cent.) occurred on the right side and fourteen (53.8 per cent.) on the left. Ten (38.5 per cent.) involved the frontal bone, and seven (26.9 per cent.) the posterior parietal region. Twenty-four (92.3 per cent.) occurred in a single cranial bone, while of the linear fractures only 41.7 per cent. were confined to one bone. Of the two remaining depressed

fractures involving two or more cranial bones, one occurred in the left temperofrontal region, and the other, a very extensive, crushing fracture, involved the left frontoparietotemporal bones. This patient developed a psychosis. The majority of these depressed fractures were compound. They have been indicated schematically in diagrams shown in Figures 5 and 6.

CONSIDERATION OF MORTALITY

Mortality.—In this series there were forty-four deaths, a mortality of 25.7 per cent. Others have reported a mortality rate varying from 14.3 per cent. in selected cases to 70 per cent. The following percentages are given from various clinics:

1. Besley[1]: 53 per cent. from 1,000 cases analyzed at the Cook County Hospital, Chicago, 1916.
2. Crandon[2]: 44 per cent. from a series of 530 cases at the Boston City Hospital.
3. Kantorowicz[3]: 29.3 per cent. in seventy-three basal skull fractures.
4. Schwarz[4]: 36 per cent. in thirty-eight cases reviewed.
5. Wanker[5]: 28 per cent. in sixty-six cases.
6. Frazier[6]: 59 per cent. from the Methodist Episcopal Hospital, Philadelphia.
7. Ransohoff[7]: 63 to 70 per cent.
8. Mixter,[8] reporting from the Massachusetts General Hospital before 1911, gave a mortality of 57 per cent. and since, a mortality of 41 per cent.
9. Sharpe[9]: Estimated the death rate in the larger New York hospitals from 1900 to 1910 as ranging from 46 to 68 per cent., and presented a mortality of 30.7 per cent. in a series of 239 cases personally observed.
10. Stewart[10]: 52.10 per cent. in 617 cases, 52.37 per cent. in nonoperative cases, and 51.7 per cent. in the operative series.
11. Brown and Strecker,[11] operative mortality, 35.2 per cent.; nonoperative 24.4 per cent. in the 100 cases studied.
12. Bower[12]: A combined mortality (operative and nonoperative) in acute cases, 26.1 per cent.

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1. Besley, F. A.: A Contribution to the Subject of Skull Fractures. J. A. M. A. **66**:345-350 (Jan. 29) 1916.
2. Crandon, L. R. G. and Wilson, L. F.: Ann. Surg. **44**:823-841, 1906.
3. Kantorowicz: Thesis: Sur. Prognose d. Schadelbases-bruche, Liepzig, 1908.
4. Schwarz: Schadelbasis-bruche, Dissertation, Goettingen, 1900.
5. Wanker, R.: Dissertation, 1900.
6. Frazier, C. H.: Problems and Procedures in Surgery, J. A. M. A. **52**:1805 (June 5) 1909.
7. Ransohoff, J.: Tr. Am. Surg. A. **28**:560-577, 1910.
8. Mixter, W. J.: Boston M. & S. J. **177**:518 (Oct. 11) 1917.
9. Sharpe, W.: Observations in the Diagnosis and Treatment of Brain Injuries in Adults. J. A. M. A. **66**:1536-1540 (May 13) 1916.
10. Stewart, J. W.: J. A. M. A. **77**:2030-2035 (Dec. 24) 1921.
11. Brown, H. P., Jr., and Strecker, E. A.: Ann. Surg. **79**:198-205 (Feb.) 1924.
12. Bower, J. O.: Ann. Surg. **78**:433 (Oct.) 1923.

It is significant that in a majority of the fatal cases, death occurs forty-eight hours after injury. Cushing[13] remarks that "the average duration of life in fatal cases is said to be forty-four hours: so there is some basis for the old rule laid down by Bergmann and Wagner that survival over two days gives a favorable prognosis." The day of death in our series agrees closely with such data in other reports; 68.1 per cent. of the fatalities occurring within the first forty-eight hours.

	Hours	Per Cent.
First day	21	47.7
Second day	9	20.4
Third day	1	2.3
Fifth day	3	6.8
Sixth day	1	2.3
Seventh day	2	4.5
Thirteenth day	1	2.3
Seventeenth day	1	2.3
Eighteenth day	1	2.3
Twentieth day	1	2.3
Thirtieth day	1	2.3
Thirty-sixth day	1	2.3
Nintieth day	1	2.3
Total	44	100.1

All deaths occurring after the seventh day were caused by a complication, either general meningitis (six), septicemia (one) or brain abscess (one). To this finding, which occasioned some surprise, there was only one exception; a patient with chronic subdural hemorrhage died five months after the injury, an occipital fracture, which will be considered separately (Case 2).

TABLE 2.—*Tabulation of Deaths from Fracture of the Skull*

Decade	Number of Deaths	Total Number of Cases	Mortality Percentage
First	1	12	8.3
Second	5	22	23.8
Third	7	31	22.6
Fourth	2	25	8.0
Fifth	12	24	50.0
Sixth	6	25	24.0
Seventh	2	12	16.7
Eighth	0	7	....
Ninth	1	8	100.0
Age unknown	8	12	66.7
	44	171	

Formerly, we were of the opinion that elderly persons withstood skull fracture poorly, while younger ones sustained the injury more satisfactorily. With this in mind, we tabulated the deaths (Table 2) according to decades and found that the highest mortality (100 per

13. Cushing, H.: Keene's Surgery, **3:82**

cent.) fell in the ninth decade—only one death, that of an old man who was struck by a train; while the fifth decade came second (50 per cent.). Those in the second and third decades of life correspond closely with those in the sixth and seventh decades (roughly sixteen, or 24 per cent.). It is a curious fact that the seven patients in the eighth decade all recovered. Obviously, in these cases, the degree of associated brain injury must have been slight.

Every patient entering the hospital with a head injury suffered from shock to a greater or less degree. With this in view we have computed as near as possible the immediate cause of death. Fay [14] and many others have pointed out that patients with head injury, dying from shock, have a low lethal temperature, a rapid respiration and a rapid thready

The type of accident causing death is shown in the following table.

TABLE 3.—*Causes of Death*

Type of Accident	Number of Deaths	Types, per Cent.	Total Number Cases	Deaths, per Cent.
Assault	6	13.6	14	42.8
Automobile (struck by)	21	47.8	83	25.3
Fall	5	11.4	25	20.0
Street car (struck by)	4	9.1	17	23.5
Train (struck by)	2	4.5	6	33.3
Motor cycle accident	0	0.0	3	0.0
Unknown	6	13.6	23	26.1
	44	100.0	171	

TABLE 4.—*Immediate Causes of Death*

Cause	Number of Cases	Per Cent.
Undoubted shock	17	38.7
Undoubted compression	9	20.4
Meningitis	6	13.6
Other complications	12	27.3
	44	100.0

pulse; those dying from medullary compression show a high lethal temperature, a slow pulse which later becomes rapid, and Cheyne-Stokes or labored respiration. Death in more than one third (38.7 per cent.) of the fatal cases was from shock—all within the first forty-eight hours. Table 4 shows the immediate cause of death.

We were surprised to find that shock caused a greater number of deaths (38.7 per cent.) than medullary compression (20.4 per cent.).

In Table 5 the total number of deaths (44) has been summarized from the standpoint of age, sex, type of accident, type of skull fracture, mental state, complications, period in hospital and necropsy findings.

14. Fay, T.: Comparative Values of Magnesium Sulphate and Sodium Chlorid, J. A. M. A. **82**:766-769 (March 8) 1924.

TABLE 5.—*Summary of Fatal Cases*

No.	Age	Sex*	Type of Accident	Type of Fracture	Mental State	Complications	Period in Hospital	Necropsy
5	82	♂	Struck by automobile...	Linear parietal left; linear temporal right	Continuous coma	Shock.....	3 hours	Linear fracture parietal left, linear fracture temporal right
12	23	♂	Fell from automobile....	Depressed frontal	Profound coma	Fractured ribs; blood in chest; fractured right arm; shock	2 hours	Frontal depressed fracture; crushed chest; mediastinal hemorrhage; fracture of right humerus, radius and ulna
18†‡	50	♂	Struck by automobile...	Old depressed occipital (five months)	Stupor increasing	Ventricle study showed lesion right frontal	36 days	Intradural hemorrhage of right frontal
19	27	♂	Assault.....	Basal	Deep coma	Bleeding nose; general bruising; bloody cerebrospinal fluid	1 day	Basal fracture across anterior and middle fossa
26	17	♂	Struck by train.....	Compound frontal and nasal bones	Increasing coma	Left eye enucleated; infected; meningitis; fracture both bones left forearm	7 days	None
27†	54	♂	Struck by automobile...	Basal	Coma	Hemorrhage, right internal capsule (needled)	2 days	Extradural hemorrhage at base
31†	21	♀	Fell three stories.....	Linear temporal right, found at operation	Coma	Edematous brain found at operation	5 days	None
32	56	♂	Fell from street car.....	Linear occipital to foramen magnum	Increasing coma	Pulse of 132 while in hospital; bloody fluid	5 days	None
38	40	♀	Fell from street car.....	Basal	Coma	Bleeding left ear.....	12 hours	Basal fracture left middle fossa
42†	11	♂	Struck by automobile...	Compound depressed parietal right	Coma	Subdural clot right; fracture left humerus	8 hours	None
44	50	♂	Unknown.....	Linear parietal right...	Increasing coma	Bloody spinal fluid; pulse 124	2 days	None
45†	?	♂	Unknown.....	Depressed comminuted frontal	Coma	Lacerated brain; loss of substance	13 days	None
46†	19	♀	Struck by automobile...	Linear parietotemporal right	Increasing coma	Meningitis; bleeding right ear	18 days	None
51	?	♂	Struck by street car.....	Linear parietotemporal left	Coma	Fracture; dislocation hip, left	12 hours	None
64†	23	♂	Struck by automobile...	Linear occopitotemporal right	Stupor increasing to coma	Fracture left tibia, fibula, and dislocation astragalus; edematous brain	3 days	None
70†	66	♂	Struck by hammer.....	Depressed frontal left	Conscious, but became comatose	Motor aphasia on admission	17 days	Subcortical hemorrhage left frontal lobe; basal hemorrhage
72†	23	♂	Struck by automobile...	Comminuted frontotemporal left	Coma	Bleeding left ear, nose and mouth; Wassermann suspicious	2 days	None
73	44	♂	Struck by automobile...	Linear occipital left....	Coma	Bleeding left ear; bloody spinal fluid	2 days	None
76	42	♂	Struck by automobile...	Separation lambdoid suture	Semicomatose increasing to coma	Bloody spinal fluid.....	2 days	None
81†	5	♀	Struck by automobile...	Compound comminuted parietal left	Coma	Laceration of brain, loss of substance; paralysis right leg	7 hours	None
85	?	♂	Struck by automobile...	Basal	Coma	Bleeding both ears and nose	20 hours	Large basal hemorrhage; edematous brain

88	62	♂	Unknown.....	Linear parietal left.....	Coma	Bleeding nose; ecchymosis neck	2 days	Linear fracture temporal left
89	45	♂	Unknown.....	Linear parietotemporal right	Coma	Fracture first ribs both sides	14 hours	Linear fracture posterior parieto-occipital right base; comminution of petrous bone
91	48	♂	Fell from automobile....	Compound, comminuted depressed temporal left	Semicoma	Fractured ribs.....	20 hours	None
94†	21	♀	Assault.....	Linear parietal right...	Conscious with increasing coma	Fractured mandible, brain abscess; operated three times	3 months	Meningitis; brain abscess, left temporal
102	?	♂	Unknown.....	Linear extensive, basal	Coma	Fractured pelvis.....	2 hours	Subdural and extradural hemorrhage; linear extensive basal
106†	?	♂	Fell on floor.....	Linear parieto-occipital left	Coma	Bleeding left ear, subdural hemorrhage left; bloody spinal fluid	7 days	Basal fracture; large subdural hemorrhage right
109†	44	♂	Struck by automobile...	Linear temporal right..	Semicoma	Compound fracture clavicle right; bleeding middle meningeal	17 hours	None
115	?	♂	Struck by automobile...	Compound occipital....	Coma	Lacerated and oozing brain	1 hour	Contusion brain; subdural hemorrhage; extensive linear fracture right occipital and basal
120	?	♂	Struck by automobile...	Depressed parietotemporal right	Coma	Extensive contusion left shoulder	2 hours	Depressed parietotemporal fracture, right
122	81	♂	Struck by automobile...	Linear frontal right....	Coma	Bleeding nose and mouth	5 days	None
124†	55	♂	Assault.....	Depressed frontal extensive	Coma	Subdural clot left, extensive	3½ hours	Extensive depressed frontal, left temporal and parietal
127	40 ?	♂	Struck by street car.....	Comminuted, depressed, posterior parietal left	Coma	Edema of lungs.....	20 hours	None
129†	36	♂	Unknown.....	Linear parietal right...	Semiconscious	Jacksonian epilepsy, left; subdural clot, right	2 days	Linear fracture across vault; no clot
136	11	♂	Assault.....	Stellate posterior parietal right	Coma	Bleeding ear right; otitis media; meningitis	30 days	Ventricles filled with pus; tuberculosis and pulmonary tuberculosis
139†	50	♂	Struck by automobile...	Linear temperoparietal left; also depressed area	Increasing coma	Bleeding left ear; extradural, subdural and subcortical clot left	6 days	None
140	50	♀	Struck by automobile...	Depressed frontal and occipital	Coma	Fracture of both femurs..	7 hours	Fracture of occiput; subdural hemorrhage
141	50	♂	Struck by automobile...	Basal	Coma	Edematous lungs.....	7 hours	Basal skull fracture; large subdural hemorrhage
145	50	♂	Struck by automobile...	Linear parietal right to ear; linear occipital to foramen magnum	Conscious, later going into coma	Bleeding both ears; otitis media right; septicemia (hemolytic streptococcus)	20 days	None
151†	12	♂	Struck by automobile...	Comminuted frontoparietal right	Coma	Extradural clot right.....	3 days	None
154	56	♂	Struck by automobile...	Linear occipital to foramen magnum	Coma	Edematous lung.....	12 hours	None
158†	40	♀	Struck by street car.....	Linear frontal left.....	Coma	Fractured metacarpals; crushed foot	2 days	None
163	20 ?	♀	Assault.....	Linear occipital to foramen magnum	Coma	General bruising.....	7 hours	None
170	27	♂	Struck by automobile...	Basal	Coma	Bleeding nose; meningitis	7 hours	Suppurative meningitis; subdural hemorrhage

* In this column, ♂ indicates male; ♀, female. † Operation performed. ‡ Special case report.

Unfortunately, we were unable to secure postmortem examination in twenty-one of these cases as they came under the coroner's jurisdiction, and in such cases necropsy is done only in cases of obscure diagnosis or suspected foul play. Twelve of these cases were complicated by fractures occurring elsewhere than the skull. Only six of this group had purely basal fractures, which were demonstrated at necropsy. All but two patients entered in a comatose or semi-comatose condition and died without improving mentally. In only one instance did the patient completely recover consciousness; this was a little girl who developed a brain abscess several weeks after her injury and died three months from the time of her admission to the hospital. In the following instance, the patient remained in a stuporous condition for many days; death was finally caused by an unsuspected subdural clot.

REPORT OF CASES

CASE 1.—V. K., a man, aged 50, married, a baker, was admitted Aug. 23, 1923 in a stuporous condition. It was stated that he had been in an automobile accident five months previously when he sustained a dislocation of the right shoulder and scalp laceration. The head injury was not considered serious, and he was discharged from the hospital two days after admission. During the following six weeks at home, he complained frequently of headache which often kept him awake at night. The headache gradually became more constant, although he attended to matters at home in the usual way. He did not vomit, was not nauseated and had no convulsions. The headache, however, became more severe; and seven days before admission he became stuporous. He was removed to another hospital where lumbar puncture revealed slightly yellow spinal fluid under 200 mm. pressure, containing two cells; the butyric acid reaction was negative, and there was a plus minus Wassermann reaction. The blood pressure was: systolic, 137; diastolic, 76; hemoglobin, 80; red blood cells, 6,000,000; white blood cells, 5,400. The differential count revealed: polymorphonuclears, 78 per cent.; large lymphocytes, 5 per cent.; small lymphocytes, 14 per cent., and transitionals, 3 per cent. There was possibly some weakness of the left side of the face, the left arm and the left leg. The eye grounds showed that both discs were blurred with overfilling, and imbedding of the veins. The pupils were small and irregular, but equal. The superficial and deep reflexes were all obtained and seemed within limits of normal and equal. No ankle clonus or Babinski's reflex was obtained on either side: no astereognosis or sensory change could be made out. The patient was unable to stand; he showed a tendency to fall backwards and to the left. There was no speech disturbance, no agraphia and no alexia. A roentgen-ray examination of the skull showed a marked depression in the mid-occipital region which gave the appearance of an old fracture. The question of diagnosis was disputed. Dr. Eliot Alden removed the depressed area in the occipital region and tapped the lateral ventricle, getting clear fluid. The patient remained under observation until Sept. 25, 1923; at times he was noisy and disturbed, and at other times he was in a rather deep stupor.

September 26, a ventricle study by air inflation showed both ventricles displaced markedly toward the left, with the right ventricle much smaller than the left and the convexity of the outer wall inward, as though there were

pressure in the right frontotemporal region. The next day the patient was noisy at times, and again stuporous. Exploration was to have been performed the next morning, but the patient died suddenly that night.

Necropsy revealed the presence of a large hematoma, apparently subdural, in the right frontotemporal region. This was surrounded by a capsule resembling the two layers of the dura. It contained greenish yellow fluid and black blood clots. The point of bleeding was assumed to be from the middle meningeal. Although the pathologist was inclined to believe that it was an intradural hemorrhage, we still feel that it was subdural with proliferation of endothelial cells, possibly from the dura, which formed its lining membrane. The condition in many respects resembled pachymeningitis hemorrhagica interna.

TABLE 6.—*Cases of Meningitis* *

No.	Age	Sex	Type of Fracture	Interval from Injury to Onset of Meningitis	Duration of Meningitis	Portal of Entry	Spinal Fluid	Organisms
26	17	♂	Compound comminuted involving frontal sinus ethymoid and cribriform regions	4 days	3 days	Left socket cribriform	Turbid	Streptococcus, pneumococcus
3	21	♀	Basal fracture	2 days	5 days	Bleeding right ear; brain abscess ?	Bloody; 130 white cells	Culture sterile ?
46	19	♀	Basal fracture	6 days	12 days	Bleeding right ear; later otitis media	Bloody; increased pressure	Pneumococcus and staphylococcus
94	21	♀	Linear right parietal; fractured mandible	10 weeks	13 weeks	Hematogenic brain abscess, left temporal	Not taken; necropsy disclosed meningitis	Culture sterile
136	11	♂	Basal fracture	15 days	15 days	Bleeding right ear; later otitis media	Not taken; necropsy disclosed meningitis	Tubercular ?
170	27	♂	Fracture extending to frontal sinus, ethymoid, etc.	3 days?	5 days	Frontal sinus, ethymoid, etc.	Pure pus	Pneumococcus
171†	32	♂	Linear parietotemporal to auditory canal; second fracture meatus	6 days	Recovered	Right ear	Pure pus	Mucosus capsulatus of Friedländer

* Percentage of deaths, 85.8 per cent.
† Special case report.

MENINGITIS CASES

Seven cases of meningitis occurred in the series, with one recovery; this will be considered separately. These have been tabulated from the standpoint of age, sex, type of fracture, interval from injury to onset of meningitis, duration of meningitis, portal of entry, spinal fluid findings and causative organisms (Table 6).

It is a striking fact that all the cases in which meningitis developed the patients were relatively young, the ages ranging from 11 to 32 years.

This gives rise to the question whether or not older persons have a greater immunity against infection of the meninges than younger ones. In three cases, the fracture was confined to the base; in three others the vault was also included. The only case in which the fracture did not extend to the base was one in which a brain abscess developed later in the temporal lobe on the opposite side. This patient subsequently died of meningitis. The shortest interval from the time of injury to the onset of meningitis was two days. The longest interval was fifteen days; this occurred in the brain abscess case referred to above. The portal of entry in four instances was through the ear; in two others it

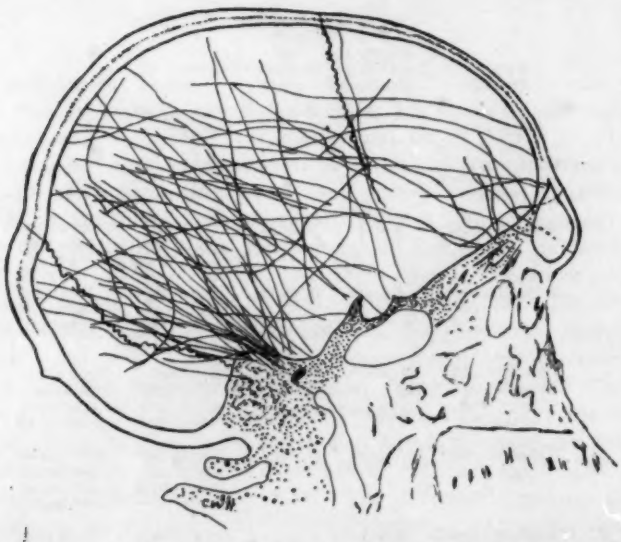

Fig. 1.—Composite tracing of linear fractures; right lateral position. The majority of the lines occur in the posterior parietotemporal region, coursing down toward the external auditory canal. Several bizarre types of fractures can be seen.

was through the upper nasal passages by way of the cribriform plate or ethmoid cells. It is to be regretted that the laboratory work on the spinal fluid was meager. In two cases no lumbar puncture was performed, the first because of the fear of spreading infection from a complicating brain abscess; the second was a case of assault. In the latter, an unexpected tuberculous meningitis was found at necropsy. Pneumococcus was present alone in one instance; in two others, it was present with staphylococcus and streptococcus, respectively. The culture was sterile in two instances. The only case in which the patient recovered showed mucosus capsulatus of Friedländer in pure culture.

Case 2.—J. K., a man, aged 32, married, a German woodchopper was admitted March 22, 1924. Nine days previous to his admission, while he was cutting trees in a wood, the patient had been struck on the right side of the head by a falling branch. He was unconscious for a short time, and bled profusely from the right ear: the bleeding gradually ceased after twenty-four hours. After that "water" drained in large quantities for another twenty-four hours, and for several days the ear was irrigated three times a day. On admission, the patient was found to be in a perfectly clear mental condition, but he complained of headache and an exquisite tenderness when any attempt was made to move the lower extremities. There was some indefinite paresthesia, such as numbness and coldness of the lower limbs. The right ear drum was perforated, and the hearing was impaired on this side. There was marked cervical rigidity and opisthotnos. Brudzinski's sign was markedly positive. The Kernig sign was positive when the feet were lifted even 6 inches (15.2 cm.) from the

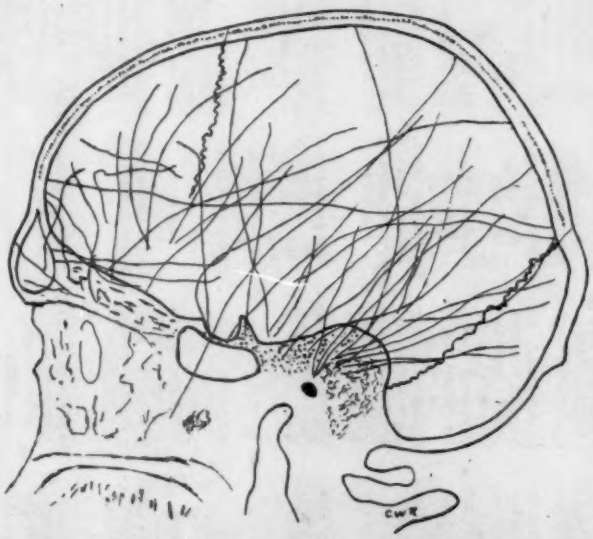

Fig. 2.—Composite tracing of linear fractures; left lateral position. Fractures on this side are much less numerous than on the right. The general course of the fracture lines is very similar to those in Figure 1.

bed. There was retention of urine. The pulse rate was 66, respirations 20 and the temperature 100 F. The examination of the spinal fluid revealed 10 c.c. of pure thick pus, which required aspiration. After that, the fluid, although thick and turbid, came spontaneously with a pressure reading of over 300 mm. The microscopic examination revealed numerous pus cells and very many small, encapsulated Gram negative bacilli of various sizes. A pure culture of *Bacillus mucosus-capsulatus* (Friendländer's) was obtained.

Immediately, 30 c.c. of one per cent. aqueous solution of mercurochrome-220 soluble was given intravenously with no clinical reaction. The pulse rate remained around 52 to 56 for twelve hours; then it went to approximately 90, where it remained. March 23, the fluid was still very purulent, showing a pressure of over 300 mm. No bacilli were found in the smears, and the culture was negative. No red tinge could be detected in the spinal fluid. The retention

TABLE 7.—*Cases in Which Operations Were Performed*

No.	Age	Sex	Mental State on Admission	Spinal Fluid	Roentgen-ray Findings	Preoperative Diagnosis	Postoperative Diagnosis	Indications for Operation	Type of Operation	Time Before Operation	Stay in Hospital	Complications	Result
1	18	♂	Semi-conscious	Bloody; 300 mm. pressure	Linear fracture right parietal	Subdural clot right	No clot; edematous brain	Increasing coma	Right subtemporal decompression; drainage	3 days	4 weeks	None	Walked out
2	15	♂	Conscious	Clear; 300 mm. pressure	Linear fracture right parieto-temporal	Subdural clot right; face center	Extradural clot right; face center	Facial convulsions left side on fifth day	Right subtemporal decompression; evacuation of clot; drainage	6 days	2 mos.	None	Walked out
3	44	♂	Semi-conscious	Bloody; 105 mm. pressure	Stellate fracture right frontal depressed	Compound depressed fracture right frontal	Compound depressed fracture right frontal; lacerated brain	Increasing coma; depressed fracture	Débridement scalp; removal fragments; drainage	1 day	2 mos.	Infected wound; local meningitis; delirious four weeks	Walked out
15	46	♂	Conscious	Not taken	Fracture left frontal, entering sinus	Depressed left frontal	Depressed left frontal	Depressed fracture left frontal	Elevation of fragments	2 hrs.	17 days	None	Walked out
18*	50	♂	Semi-conscious	Clear	Depressed fracture occipital; ventricles displaced to left	Depressed fracture occipital old; brain tumor	Depressed fracture occipital; intradural hemorrhage; right temperofrontal	Increasing coma, choked disks; old depressed fracture	1. Removal depressed fragments; 2. Ventriculography	1. 11 days 2. 35 days	36 days	Hyperpyrexia and medullary compression	Died; necropsy, extensive intradural hemorrhage right temperofrontal, evidently old
21	34	♂	Conscious	Bloody; 270 mm. pressure	Compressed fracture left posterior parietal	Depressed fracture left parietal	Depressed fracture left parietal; laceration of brain	Depressed fracture; increasing headache; increasing active deep reflexes, right	Removal of fragments, drainage	3 days	15 days	None	Walked out
22	27	♂	Semi-conscious	Bloody; 300 mm. pressure	Depressed fracture right posterior parietal	Depressed fracture right posterior parietal	Depressed fracture right posterior parietal; dura under increased tension	Depressed fracture	Removal of depressed fragments; incision dura; drainage	10 hrs.	3 days	None	Transferred
24	8	♀	Semi-conscious	Not taken	Extensive bursting fracture right occipitoparietal	Fractured skull right concussion; subdural clot	Fractured skull right macerated brain	Increasing coma, bradycardia	Right subtemporal decompression; removal macerated brain substance; drainage	1 day	19 days	None	Walked out
27	54	♂	Coma	Bloody	Linear fracture right temporo-parietal	Linear fracture right parietal; subdural clot	Edematous brain; intracapsular hemorrhage, right	Left hemiplegia	Right subtemporal decompression; partial evacuation of clot, right internal capsule	9 hrs.	2 days	Cerebral anemia, compression	Died; necropsy, basal hemorrhage, extradural; basal skull fracture
28	55	♂	Coma	Bloody	Linear fracture right parieto-temporal	Subdural clot left parietal	Linear fracture right parietal; subdural clot left	Subdural clot left parietal; increasing coma; increasing deep reflexes, right	Left subtemporal decompression; evacuation of clot; drainage	3 days	18 days	Subdural clot contracoup to linear fracture	Walked out

* Special case report.

51	21	♀	Coma	Bloody	None taken	Edematous brain; basal fracture	Edematous brain; linear fracture, right temporal	Coma; choked discs	Right subtemporal decompression; drainage	1 day	5 days	Blood Wassermann test, four plus; meningitis ?	Died; no necropsy
42	11	♂	Coma	Not taken	Extensive linear fracture right parietotemporal, with separate fragment right parietal	Comminuted fracture right frontoparietal; right subdural hemorrhage	Comminuted fracture right frontoparietal; right subdural hemorrhage	Comminuted fracture right parietal; subdural clot right	Right subtemporal decompression; evacuation subdural clot; drainage	2 hrs.	8 hrs.	Shock	Died; no necropsy
45	?	♂	Semi-conscious	Not taken	Depressed comminuted fracture frontal involving sinus	Depressed fracture frontal involving sinus; concussion and contusion	Depressed fracture frontal involving sinus; concussion and contusion	Depressed fracture frontal	Depressed fracture elevated; removal macerated brain substance	1 hour	18 days	Macerated brain	Died; no necropsy
46	19	♀	Semi-conscious	Bloody; increased pressure	Linear fracture right parieto-temporal	Linear fracture right temporal region	Depressed fracture right temporal region; lacerated brain	Extreme restlessness and semi-coma persisting	Right subtemporal depression, macerated brain; removal displaced fragments; drainage	3 days	18 days	Meningitis; infected wound; bleeding from nose and right ear	Died; no necropsy
56	16	♂	Irrational	Not taken	Depressed fracture frontal bone and frontal sinus	Depressed fracture frontal region, compound	Depressed fracture frontal region, compound	Depressed compound fracture forehead	Elevation of fracture; removal of some fragments; drainage	3 hrs.	1 day	None	Transferred; ultimately well
64	28	♂	Semi-conscious	Bloody	Linear fracture right occipito-temporal	Linear fracture right occipito-temporal; increased intracranial pressure	Increased intracranial pressure; linear fracture right occipito-temporal region	Edema of brain; coma	Right subtemporal decompression; drainage	2 days	3 days	Fracture left tibia and fibula; dislocation astragalus	Died; no necropsy
65	24	♂	Semi-coma	Not taken	Linear fracture occipital	Linear fracture occipital; increased intracranial pressure	Increased intracranial pressure; linear fracture occipital	Increasing coma	Right subtemporal decompression; drainage	2 days	49 days	Wassermann, four plus	Walked out
69	28	♀	Semi-conscious	Not taken	Linear fracture occipital	Subdural clot left motor area	Subdural hemorrhage left motor area	Epilepsy, Jacksonian right; motor aphasia	Bone flap left motor area; removal clot	1 day	15 days	Wassermann, four plus	Transferred
73	23	♂	Coma	Not taken	Multiple fracture left fronto-temporal	Depressed fracture left frontal	Compound depressed fracture left frontal	Compound depressed frontal fracture	Elevation and removal of fragments; drainage	2 hrs.	2 days	Wassermann, "suspicious"	Died; no necropsy
81	5	♀	Coma	Not taken	Not taken	Compound comminuted fracture left parietal; oozing brain	Compound comminuted fracture left parietal; oozing brain	Compound comminuted fracture, left parietal; oozing brain	Removal fragments and macerated brain substance drainage	3 hrs.	7 hrs.	Shock	Died; no necropsy
94	21	♀	Conscious	Not taken	Linear fracture right parietal	Linear fracture right parietal; brain abscess left	Linear fracture right parietal; brain abscess left	Signs and symptoms of brain abscess left side	Drainage of abscess on three occasions	37 days	90 days	Spreading of abscess; meningitis	Died; necropsy showed multiple abscesses left hemisphere and general meningitis

TABLE 7.—*Cases in Which Operations Were Performed—(Continued)*

No.	Age	Sex	Mental State on Admission	Spinal Fluid	Roentgen-ray Findings	Preoperative Diagnosis	Postoperative Diagnosis	Indications for Operation	Type of Operation	Time Before Operation	Stay in Hospital	Complications	Result
101	43	♂	Coma	Not taken	Old fracture left frontal with defect	Old comminuted fracture, left frontal; new basal skull fracture	Old comminuted fracture, left frontal; new basal skull fracture	Jacksonian epilepsy; headache; frontal skull defect	Repair of skull defect; left frontal	16 days	39 days	Epilepsy, Jacksonian, right; Wassermann, four plus; visual hallucinations	Walked out
108	?	♂	Coma	Not taken	Linear fracture left parieto-occipital	Subdural hemorrhage left; linear fracture left parieto-occipital	Subdural hemorrhage left; linear fracture left parieto-occipital	Subdural clot, left	Left subtemporal decompression; removal of subdural clot	4 days	7 days	Bleeding nose and left ear; hyperpyrexia at death	Basal fracture across middle fossa extending bilaterally to occiput; subdural clot right temporal
109	49	♂	Semi-conscious	Bloody; normal pressure	Linear fracture right temporal	Middle meningeal hemorrhage, right; linear fracture right temporal	Subdural clot right; bleeding meningeal; depressed fracture right temporal	Bleeding middle meningeal	Right subtemporal decompression; bleeding middle meningeal tied	7 hrs.	17 hrs.	Shock	Died; no necropsy
124	55	♂	Coma	Not taken	Not taken	Depressed fracture left temporal; subdural clot	Depressed fracture, left temporal; subdural clot; also extradural clot	Extensive depressed fracture left temporal	Left craniotomy elevation and removal of fragments; evacuation extra and subdural clots; drainage	2½ hrs.	3⅓ hrs.	Shock	Died; necropsy showed extensive linear fracture left temporal with overlapping of upper fragment; no clot
129	36	♂	Semi-conscious	Not taken	Linear fracture horizontal, right parietal	Linear fracture right parietal; subdural clot; Jacksonian epilepsy left	Linear fracture right parietal; subdural clot; Jacksonian epilepsy left	Jacksonian convulsions left	Right subtemporal decompression; evacuation subdural clot; drainage	29 hrs.	2 days	Shock	Died; necropsy showed fracture running across top of skull through both parietal bones; no clot
132	11	♂	Conscious	Not taken	Depressed fracture left cerebellar floor	Depressed fracture left cerebellar floor; old linear fracture right frontal	Depressed fracture left cerebellar floor; old linear fracture right frontal	Left sided cerebellar symptoms; i. e., ataxia, nystagmus, headache, restlessness, vertigo, etc.	One fragment removed, other fragments elevated; dura not opened	4 hrs.	36 days	None	Walked out
139	50	♂	Semi-conscious	Bloody; 300 mm. pressure	Linear fracture left temporo-parietal; irregularity which may be depressed	Linear fracture left; subdural clot	Linear and depressed fracture left; extradural, subdural and subcortical clot	Compound fracture left; depressed; subdural clot	Left subtemporal decompression; removal of extradural clot; subcortical clot ruptured spontaneously; drainage	2 days	6 days	Bleeding left ear; rigid neck; contused brain	Died; no necropsy

143	20	♂	Coma	Not taken	Stellate fracture; right posterior parietal depressed	Left sided convulsions coma; depressed fracture right parietal	Depressed fracture right parietal	Jacksonian convulsions left; depressed skull fracture	1. Right subtemporal decompression; drainage. 2. Elevation depressed fracture; evacuation extra and subdural clots; drainage	1. 1 day 2. 4 days	43 days	Fracture explored at outside hospital; depression not appreciated; subtemporal decompression done 3 days before elevation of fracture at second stage	Walked out
151	12	♂	Coma	Not taken	Comminuted fracture of vault, frontal and right parietal	Left sided epilepsy; coma	Left sided epilepsy; coma	Jacksonian epilepsy left	Right exploratory craniotomy; removal extradural clot	3 hrs.	29 hrs.	Shock; 5% sugar in urine	Died; no necropsy
159	24	♂	Conscious	Not taken	Depressed fracture frontal sinus	Depressed fracture frontal sinus	Depressed fracture frontal sinus	Depressed fracture frontal sinus	Elevation of depressed fragments	6 days	12 days	None	Walked out
102	39	♂	Semi-conscious	Not taken	Area of bone loss, left parietal	Depressed fracture left parietal	Depressed fracture left parietal	Depressed fracture left parietal	Removal of depressed fragments left parietal	2 hrs.	14 days	None	Walked out
111	11	♀	Conscious	Not taken	Linear fracture right frontal, temporal and parietal	Linear fracture right parietal, etc.; edema of brain	Linear fracture right parietal, etc.; edema of brain	Increasing coma and bradycardia	Right subtemporal decompression; drainage	2 days	19 days	None	Walked out
70	66	♂	Conscious	Not taken	Circular depressed fracture, left frontal	Depressed fracture left frontal; motor aphasia	Depressed fracture left frontal; motor aphasia	Depressed fracture left; motor area	Elevation of depressed fragment; removal of extradural clot	6 days	17 days	Softening of brain	Died; necropsy showed cortical and subcortical hemorrhage left motor area; subdural basal hemorrhage; contused brain
112	24	♂	Conscious	Not taken	Depressed fracture, left frontal	Depressed fracture left frontal	Depressed fracture left frontal	Compound depressed fracture left frontal	Removal of fragments	2 hrs.	24 days	None	Walked out
158	10	♀	Coma	Not taken	Comminuted fracture left frontal	Compound, comminuted depressed left frontal	Compound, comminuted depressed fracture left frontal	Compound comminuted left frontal	Exploratory craniotomy and elevation of fragments	2 hrs.	2 days	Crushed foot; shock	Died; no necropsy
168	33	♂	.....	Not taken	Comminuted fracture, right frontal	Compound depressed right frontal	Compound depressed right frontal	Compound depressed right frontal	Elevation of fragments	2 hrs.	7 days	None	Walked out
152	63	♀	Coma	Not taken	Linear fracture left frontal	Fracture left frontal, extradural clot left	Fracture left frontal; subdural clot left	Linear fracture left frontal and temporal	Left subtemporal decompression; evacuation of subdural clot	6 hrs.	18 days	None	Walked out

of urine had disappeared. On March 24, a second intravenous injection of mercurochrome-220 soluble was given without clinical reaction. March 25, the rigidity of the neck was much diminished, and the Kernig sign was less marked. On March 26, the third intravenous injection of mercurochrome-220 soluble was given simultaneously with the spinal puncture. The fluid was slightly turbid but not red tinged. The pressure was 120 mm. By March 27, the rigidity of the neck was entirely gone. The Kernig sign was still present but much diminished. Both urine and stool were stained red by mercurochrome. On March 28, the fourth treatment by intravenous injection of mercurochrome-220 soluble

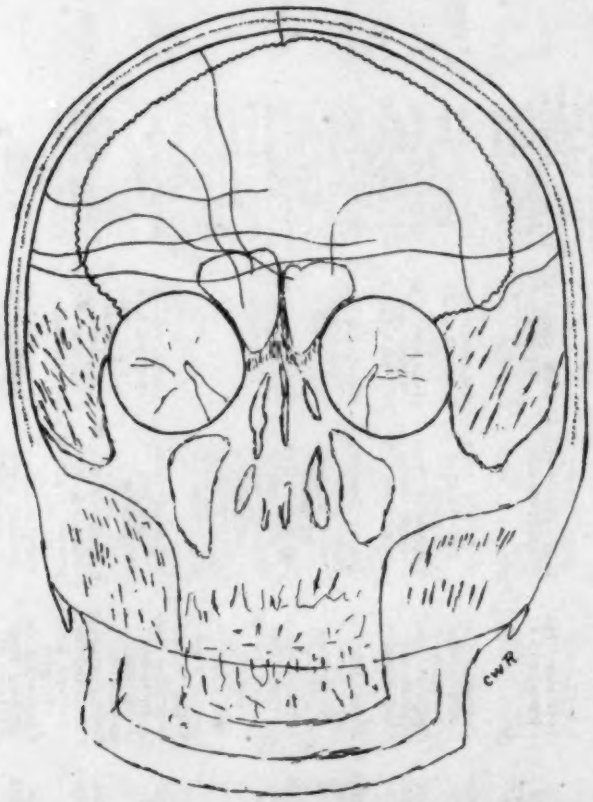

Fig. 3.—Seven linear fractures; frontal position. Only two are confined to the frontal bone. The peculiar and extensive course of the remaining five may be noted.

was given with simultaneous lumbar puncture. The spinal fluid was perfectly clear, although a pedicle formation was noted after it had stood two hours. The cell count was fourteen; globulin, two plus and the sugar content, 0.7 per cent. April 2, the spinal fluid was found to be perfectly clear and under normal pressure. The cell count was three per cubic millimeter, with a negative globulin reaction. The patient's temperature and pulse were normal at that time, and he felt quite well. The Kernig sign had now entirely disappeared, and he left the hospital April 9.

CASES IN WHICH OPERATIONS WERE PERFORMED

In thirty-eight cases (22.2 per cent.) operations were performed. These cases have been tabulated from the standpoint of age, sex, mental state on admission, spinal fluid findings, roentgen-ray findings, preoperative diagnosis, postoperative diagnosis, indications for operation, type of operation, stay in hospital, complications and results.

The operative mortality for these cases was considerably higher than for the entire series. Eighteen of the thirty-eight patients operated on died, giving an operative mortality of 47.4 per cent. In spite of the fact that many of these were in a critical condition, intervention was felt to be imperative because it was thought that they had either depressed fracture (often compound) or subdural clot. Sixteen patients (43.3 per cent.) of this series were thought to have depressed fracture, but eventually eighteen were proved; of these, seven died, giving a mortality of 38.8 per cent. in the cases of depressed fracture. Eleven were found to have intracranial hemorrhage of some type; of these eight died, giving a mortality of 72.7 per cent. By far the highest mortality was reached in these acute bleeding cases. In two instances depressed fracture and intracranial hemorrhage existed. Death in the remaining five cases was caused by edema and contusion of the brain, giving a mortality of 27.7 per cent. This corresponds closely to the mortality (25.7 per cent.) for the entire series including cases in which operations were performed as well as those in which there was no operation. The roentgen-ray and the operative diagnosis for depressed fracture agreed in sixteen cases. Two fractures diagnosed by the roentgen ray as linear were found at operation to be depressed. All fractures diagnosed as depressed before operation were confirmed at the table. We were mistaken in diagnosing subdural clot in two instances; in one none was encountered, and in the second the clot was extradural. In one instance where subdural clot was diagnosed before operation, extradural, subdural and subcortical clots were disclosed. In two instances extradural clot was diagnosed before operation, and a subdural clot was found. We feel that in some of the fatal cases the patients were operated on too soon, and during the last year it has been a rare occurrence for a patient with a head injury to be subjected to operation before twenty-four hours have elapsed. One case of depressed fracture of the left cerebellar floor seems worthy of special mention.

Case 3.—W. J., a boy, aged 11, was admitted March 31, 1923. On March 30, he fell from a street car. He was seen either to fall or jump from the steps, striking his head against a telegraph pole; this threw him back against the car and "the steps of the street car hit him on the top of the head," causing him to become unconscious.

When the patient was seen April 1, his mind was perfectly clear, but he was very irritable and at times drowsy. There was a laceration of the scalp

extending about 2 inches (5 cm.) over the right parietal region and 1 inch (2.5 cm.) over the left occipital region. There were brush burns on both cheeks. A small puncture wound was noted in the left sub-occipital region about 4 cm, below the external occipital protuberance and about 2.5 cm. to the left of the midline. The general physical examination was negative. There were no palsies of any extremity. The pupils were of medium size and reacted to light and distance. The nasal margins of the disks were somewhat hazy. The vessels were rather full and pulsated; otherwise the retinal structures were normal. He complained of some headache, but was not nauseated and did not vomit. He preferred to lie on the right side, and any attempt to turn on the left caused dizziness and nausea. There was a wide ataxia of the left

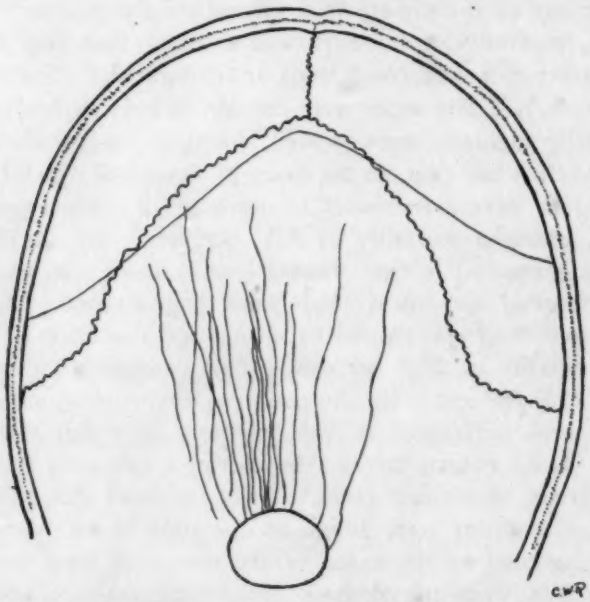

Fig. 4.—Fifteen fractures; occipital position. The uniformity with which linear fractures course down toward the foramen magnum may be noted. Of the fifteen fractures shown, ten appear from roentgen-ray studies to enter the foramen magnum. The long fracture running below the junction of the lambdoidal and saggital sutures and extending laterally was drawn from description at necropsy.

arm and the left leg. No astereognosis or sensory change was made out on either the right or the left. When he looked to the left there was marked lateral nystagmus, but there was none when he looked to the right. The biceps, triceps and knee jerks were about normal and equal. No Babinski's reflex or ankle clonus was obtained on either side. The roentgen-ray examination of the skull revealed a depressed fracture in the left suboccipital region pushing up into the posterior fossa about 2.5 cm. and compressing the left cerebellar lobe.

On April 3, a half cross bow incision was made over the left occipital region. The exposed fracture consisted of a depression measuring approximately 3 cm. in diameter and 2 cm. in depth. One fragment of bone was removed, and the rest were easily replaced. The dura was found to be bluish and bruised, but it was not opened. The wound was closed in layers without drainage. May 8,

the patient walked out of the hospital. At that time the ataxia of the left upper and lower extremities had disappeared.

June 19, the patient returned for a check-up examination. The wound was found to be well healed, and the neck movements were normal and free. The patient stated that he had had an occasional headache. The cranial nerves were normal, and there was no nystagmus in any direction. The pupils were equal and reacted to light and accommodation. Aside from a slight blurring of the nasal margin of the left disk, the eye grounds were normal; coordination was excellent for all tests. The Romberg test was negative, the patient standing on either foot alone. The gait was normal. All deep and superficial reflexes were present and normal, and no abnormal reflexes were obtained.

DIAGNOSIS AND TREATMENT

The general signs and symptoms of concussion, contusion and compression of the brain are familiar, and a review is not necessary. Opinion is divided, of course, as to whether cases with symptoms of increased intracranial pressure should be decompressed. Cases of head injury, we feel, cannot be treated collectively; each must be considered according to its individual symptoms. We have not decompressed over 15 per cent. of the cases for relief of pressure. In a number of cases in which operation was not performed there was a fatal termination within the first twenty-four or forty-eight hours; such cases naturally give rise to the question whether or not surgical intervention might have changed the course of events. We feel that in cases of depressed fracture and hemorrhage exploration should be made if the general physical condition of the patient justifies this procedure. The following items are worthy of consideration:

1. The element of shock must be considered and combated before operative procedure of any kind is instituted.

2. Careful examination, both physical and neurologic, is important and should be repeated from time to time. Roentgenograms of the skull should be taken in every case of suspected fracture. In many instances, especially in cases of basal fracture, the line will not be shown. On the other hand, when a linear fracture of the vault or base is shown, this fact should not be taken as a deciding factor for operation. Whenever possible, fractures of the skull should be demonstrated, just as fractures of the long bones are demonstrated. Pulse, temperature, respiration and blood pressure records should be made frequently. Our custom has been to have the pulse rate taken every half hour and the blood pressure at intervals of from one to two hours in severe cases. It is only by watching closely the variation in these phenomena that one can judge the opportune time for operation.

3. Ophthalmoscopic examination is required and often may be repeated advantageously. We do not feel that the eye ground picture in itself is a reliable early guide to the degree of change in intracranial

pressure. This is especially true in the first few hours after injury. Our experience includes cases terminating fatally, in which the eye grounds have shown little more than overfilling of the vessels and hyperemia of the retina. Even with the patient dying shortly after the injury, the disk margins have remained essentially clear cut, and the optic cup has been well seen. After forty-eight hours when the brain has had time to adjust itself to the condition of increased pressure, the eye ground picture becomes more significant. In one instance, a patient with a fractured skull presenting a choked disk of three diopters swelling was up and about and complaining of very little headache. The neurologic examination in this case was essentially negative.

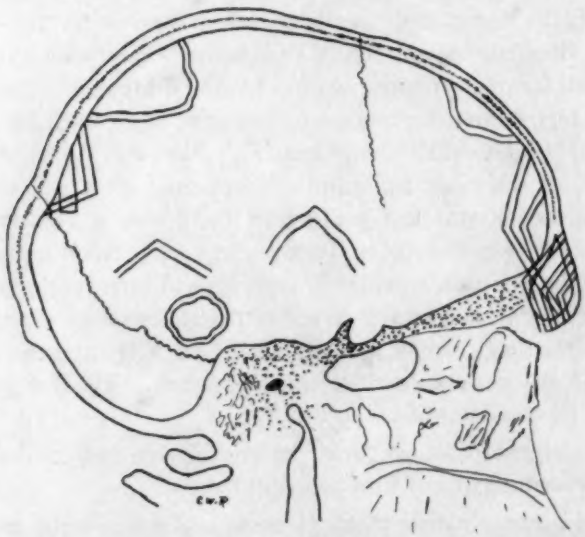

Fig. 5.—Diagramatic sketch of depressed fractures occurring on the right side; right lateral position. The location of the depression has been carefully indicated, although the exact character has not been carried out. Six of the twelve fractures shown are frontal.

4. The mental state of the patient is regarded as a fair index of his condition. This applies principally to the cases in which the patient is suffering from edema of the brain. We have seldom allowed a patient to remain unconscious for more than two or three days without doing a subtemporal decompression with drainage. On occasion, we have resorted to decompression when the patient was exceedingly irritable and disoriented, provided lumbar punctures and dehydration had failed to give relief. Unless something can be done fairly early to re-establish normal processes, we find that these patients not infrequently develop post-traumatic neurosis.

5. Examination of the spinal fluid, while not routine, is made in all cases where there is a possibility of more than simple concussion. Bloody cerebrospinal fluid was found in 85 per cent. of the cases punctured. We believe that the spinal fluid pressure readings are of more value than the ophthalmoscopic findings during the first few hours. Spinal fluid drainage is often resorted to as a therapeutic measure, especially in cases in which the pressure is increased. We have come to believe that cases of severe headache indicating increased spinal fluid pressure that is relieved by the first puncture may be much benefited if this procedure is repeated. In fact, such patients not infrequently ask to have this done. Pressure readings should be taken at each puncture. Many cases in which bloody spinal fluid is revealed, especially when it

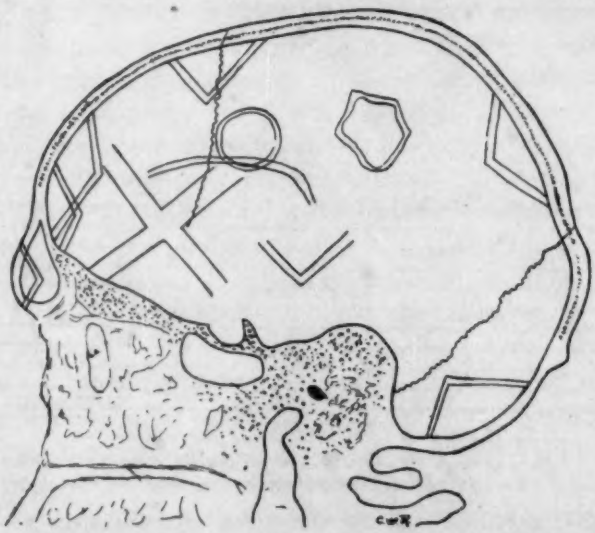

Fig. 6.—Depressed fractures occurring on the left side; left lateral position. Two cases described from necropsy findings are not included. Especial attention is called to the depressed fracture in the suboccipital region (Case 1).

is under increased pressure, present signs and symptoms of a meningitis; this disappears after the blood has been absorbed or drawn off by repeated punctures. While we have had no untoward results from lumbar puncture, we always watch the pulse and general condition of the patient while the fluid is being removed, and invariably stop if the pulse shows much variation either up or down. A cistern puncture was done in two instances only; in each case the patient was moribund on admission, and showed definite signs of medullary compression. Bloody spinal fluid was removed from the cistern in both cases, but without benefit to the patient. In spite of the impunity with which cistern puncture is now being employed for many conditions, we cannot yet bring

ourselves to believe that it is done without danger in cases presenting increased intracranial pressure from trauma.

6. Subtemporal decompression, when it is performed for relief of pressure, is usually drained. We cannot conceive of getting sufficient mechanical relief from decompression alone to accommodate an edematous brain. The drainage which usually follows during the first forty-eight hours is of great volume, as judged by the amount of fluid absorbed by the dressings; we believe it is this drainage which is largely responsible for relief. The drain is never left in over forty-eight hours. On only one or two occasions have we had an infection of the wound as a result of drainage. If the brain is found dry at operation, we sometimes close the wound tight.

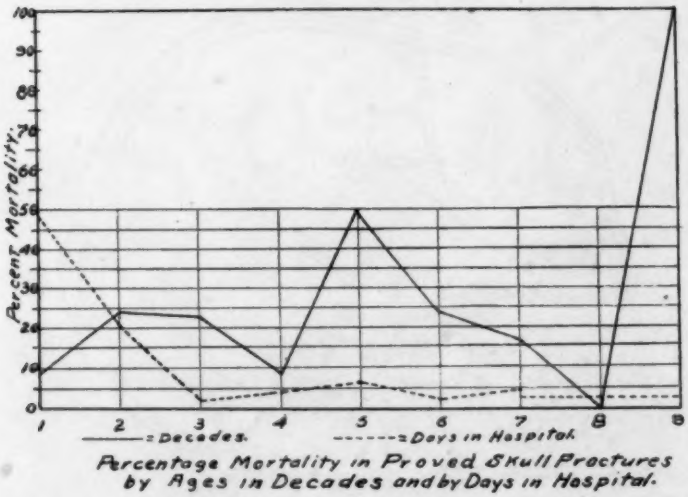

Fig. 7.—The mortality from the standpoint of the number of days after injury and of decades.

7. It has been our practice to operate in all cases of suspected local hemorrhage, for we believe that the mechanical removal of the clot is safer than absorption. For example, in one case in which a subdural hemorrhage was allowed to absorb, the patient returned six months later with Jacksonian epilepsy. At subsequent operation, the cortex was yellowish in color, and many adhesions were present at the site of the old hemorrhage.

8. It has been our practice to open the dura in the majority of cases in which an extradural clot is encountered. On one occasion we found an extradural, a subdural and a subcortical clot in the same general location. After the successful removal of an extradural clot, it is most discouraging to find that later the patient's condition was unimproved

or even worse, and, upon reopening the wound and laying back the dura, to discover a subdural clot which had not been disclosed at the first procedure. The opening of the dura may add slightly to the risk of infection, but a wide opening is not necessary in order to investigate the subdural space and may be easily closed.

9. Local anesthesia is frequently employed. If the patient is in deep stupor or coma the operation may be performed without difficulty. In cases presenting irritability and restlessness, a combined local and general anesthesia is employed, a minimum of ether being used. Large, depressed fractures have been elevated under local anesthesia when the patient was conscious throughout the procedure. There is little pain except when tension is brought to bear by leverage on the fragments. We would emphasize the importance of saving all possible fragments when a depressed fracture is being corrected. It is seldom necessary to remove all the loose fragments, and in the majority of cases most of them can be replaced satisfactorily. If great care is exercised with the dressings and bandaging for the first few days, the area soon stiffens and subsequently becomes solid. It has been our custom to save the fragments even in cases of compound fracture. Focal epilepsy following head injury is usually looked on as an indication for operation.

10. Of the 171 proved fractures, 22.2 per cent. of the patients were operated on. This group, it will be remembered, includes only proved cases and has been selected from the 580 cases admitted to the hospital as "skull fracture." The percentage of cases in which operation was performed for the entire series obviously would be considerably smaller.

11. Hypertonic saline (from 15 to 25 per cent.) has been employed intravenously in a number of cases to reduce intracranial pressure. We have not been favorably impressed with the results and have come to rely chiefly on dehydration by the administration of magnesium sulphate either by mouth or rectum as advised by Frazier. From one to one and a half ounces (28.35 to 42.52 gm.) of magnesium sulphate are given by mouth once or twice a day when it is possible, or from one to three ounces (28.35 to 85.05 gm.) of crystals dissolved in six ounces (170.1 gm.) of water are given as a retention enema every three or four hours, according to the degree of the increased intracranial pressure. It appears that more prompt relief is obtained from repeated lumbar puncture than from other methods.

SUMMARY AND CONCLUSIONS

1. One hundred and seventy-one cases of proved skull fracture are analyzed, with a mortality of 25.7 per cent. Thirty-eight patients (22.2 per cent.) were operated on, with an operative mortality of 47.7 per cent.

2. Linear fractures occurred 145 times; on the right side ninety-three times, and on the left side, fifty-two. The most common site of linear fracture was from the posterior parietal region coursing downward and forward through the temporal bone to the neighborhood of the external auditory meatus. Composite tracings of these fractures have been made.

3. Depressed fractures occurred twenty-six times; on the right side, twelve, on the left side, fourteen times. The most common location was the frontal region; second in frequency was the parietal region.

4. Sixty-eight per cent. of the deaths occurred within forty-eight hours after injury. All the deaths after the fifth day were due to complications, viz., septicemia, brain abscess or meningitis, with exceptionally a subdural hemorrhage of many months' standing.

6. In one case of proved meningitis (mucosus capsulatus of Friedländer), the patient recovered after four intravenous injections of 30 c.c. of one per cent. mercurochrome-220 soluble and repeated lumbar punctures.

7. Decompression for the relief of pressure was seldom performed under forty-eight hours.

8. A bleeding ear was never irrigated; the canal was plugged with sterile cotton.

9. Cases decompressed for relief of pressure were usually drained for forty-eight hours. We believe that the benefits obtained from drainage of cerebrospinal fluid outweigh the added risk of infection.

10. In cases of depressed fracture and of clot, the patients should be operated on if an operation is warranted by the patient's condition.

11. When an extradural clot is encountered, the dura should be opened to ascertain if a subdural clot exists.

12. We believe that the spinal fluid pressure is a more accurate early index of the degree of increased intracranial pressure than the ophthalmoscopic picture.

13. Dehydration with magnesium sulphate administered either by mouth or rectum is valuable in compression cases.

BENIGN STRICTURE OF THE BILE DUCTS*

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Unfortunately, benign stricture of the bile ducts is no longer a surgical curiosity, many cases being encountered in large surgical clinics. It therefore seems desirable to present a collective review of the experience relative to this very formidable lesion at the Mayo Clinic.

ANATOMY

The major bile duct lies in a sheath formed by the right free border of the gastrohepatic omentum. The right and left hepatic ducts unite to form the main hepatic duct, which is about 2.5 cm. in length, and lies almost wholly within the portal fissure. The common duct, formed by the union of the cystic and hepatic ducts, is a little more than 7.5 cm. in length, runs in the free edge of the gastrohepatic omentum with the hepatic artery to the left and the portal vein behind, and empties into the second part of the duodenum about 7.5 cm. from the pylorus. It has three divisions. The supraduodenal portion is from 3.2 to 3.8 cm. in length, and extends from its origin to the posterior surface of the duodenum. It is the most accessible part and can be palpated by a finger in the foramen of Winslow. The retroduodenal and transduodenal are the two other divisions. Lesions of the supraduodenal portion are probably the most important from the functional, the most difficult from the technical, and the most hazardous from the prognostic standpoint of all lesions that the surgeon has to treat. These lesions comprise any structural change that interferes with the free passage of bile through the lumen of the duct.

STRICTURE OF THE BILE DUCTS

If one judges by the literature on biliary surgery, the incidence of stricture of the bile ducts is very low. Individual reports are few and are confined to one or two cases in which operation has been successful. Undoubtedly, there are many more, particularly in recent years when cholecystectomy has been more widely practiced. Details of one or two cases may be very instructive, but rarely will be duplicated in the experience of a single surgeon. In an admirable paper by Eliot,[1] written in

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* From the division of surgery, Mayo Clinic.

1. Eliot, E.: The Repair and Reconstruction of the Hepatic and Common Bile Ducts, Surg., Gynec. & Obst. **26**:81-102 (Jan.) 1918.

1918, will be found a careful review of all cases reported until that time. The records of the Mayo Clinic contain the histories of forty-eight patients operated on for benign stricture of the bile ducts. There are two classes of benign stricture, the congenital and the acquired.

The Congenital Stricture.—The congenital variety manifests itself at, or soon after, birth by the cardinal symptoms of total biliary obstruction. Occasionally, a patient may live until adolescence, as, for example, Treves'[2] patient, who was operated on at the age of 19 because of jaundice, which had existed since the patient was 3 years old. A congenital stricture at the lower end of the common duct was found. Treves made a lateral anastomosis between the dilated portion of the common duct and the intestine, and the patient recovered. Mathieu[3] has noted twenty reported cases of congenital stricture. He believes the most common cause to be an angiocholitis occurring during intra-uterine life; and Rolleston[4] maintains that there is a primary mixed cirrhosis of the liver of the fetus, due to toxins from the maternal blood, with a descending obliterative cholangeitis that involves the extrahepatic ducts in a process similar to obliterative appendicitis. The practical points relative to congenital stricture are: (1) The condition becomes manifest in early life by permanent jaundice; there is often a cystic tumor in the right hypochondrium; (2) operation discloses enormous dilatation of the major bile ducts with the site of stricture usually at the lower end of the common duct, and (3) relief is afforded by lateral anastomosis of the common duct with the duodenum. Our records show one case of congenital stricture as follows:

REPORT OF CASE

CASE 1.—A woman, aged 28, had had a cholecystostomy when 15 because of jaundice. There was a recurrence of the jaundice eight years later, and at operation, at the Mayo Clinic, the common duct was found to be enormously dilated because of a stricture at the lower end, which contained several stones. A lateral anastomosis was made between the duct and the duodenum, but death occurred four days later.

Rolleston and others have reported congenital strictures of the common duct due to syphilis.

Acquired Stricture.—The acquired type of stricture may be inflammatory or traumatic.

The rôle of inflammation in causing stricture of the bile ducts has been compared to the same process in the urethra following gonorrheal

2. Treves, quoted by Rolleston (Footnote 4, p. 640).

3. Mathieu, Paul: Rétrécissements non néoplasiques des voies biliaires principales, Rev. de chir. **37**:61-89, 174-207, 1908.

4. Rolleston, H. D.: Diseases of the Liver, Gallbladder and Bile Ducts, London, Macmillan Company, Ltd., 914, pp. 649-663.

urethritis; but the extreme rarity of stricture of the common duct, in spite of the great frequency of inflammation, as seen at operation, impresses one with the peculiar resistance of the bile ducts to cicatricial stenosis. We believe that rarely the "fretting" of a stone in the lumen of the duct leads to localized ulcerations of the walls and, finally, to stricture. A stone may be impacted in the cystic duct or in the pelvis of the gallbladder, which is adherent to the common duct, by extension of the inflammatory process. Stricture of the common duct may be a sequel of this condition. Suppuration in the gallbladder with gangrene of its walls may involve portions of the common and hepatic ducts. Again, the introduction of gauze drains down to the duct or severe infection following cholecystectomy, as in the case reported by Brewer,[5] may terminate in destruction of a large part of the duct.

Traumatic stricture results from injury to the hepatic or common duct during cholecystectomy. It is impossible to ascertain the percentage of strictures caused by injury to the duct because, when this operation is performed, the evidence rarely affords a clue to the etiologic factor. Many competent surgeons have reported cases of traumatic injury and division of the ducts followed by immediate repair. The apparent rarity of the condition is due possibly to two factors: injuries that are not recognized, and the unusual facility with which the common duct repairs its injuries. It has been observed in experimental work on dogs that the common duct can be tied, and if the ligature is not too heavy, it will in time cut through, and the normal course of the bile be reestablished. Sullivan[6] noted that he could completely sever the common duct in the dog, and that, in the majority of instances, it would reunite spontaneously. Slight injury to the duct undoubtedly occurs with relative frequency and the fibrosis of repair causes some narrowing of the duct, but the lumen remains sufficient for the free passage of bile, and symptoms do not occur.

This brings up the question of the degree of cicatricial contraction necessary to produce obstruction. It must be very variable, and is probably subject to the same influences as in stricture of the male urethra. Jaundice is the only reliable symptom of biliary obstruction, but it seems probable that hepatic function could be impaired even to the degree of early cirrhosis by an impeded flow of bile, without producing jaundice. The chief symptoms, in many of our cases of stricture, were attacks of hepatic colic followed by jaundice, with intervening periods with complete absence of symptoms. Opening the common duct for the purpose of

5. Brewer, G. E.: Hepaticoduodenal Anastomosis, Tr. Am. S. A. **28**:308-311, 1910.

6. Sullivan, A. G.: Reconstruction of the Bile Ducts, J. A. M. A. **53**:774-777, 1909; ibid. **58**:2026-2029, 1912.

exploration or drainage is widely practiced, but, so far as we can determine, does not result in stricture. The reason commonly given is the fact that the incision is made in the long axis of the duct, but there is no proof that a transverse incision may not be equally harmless. The T tube for drainage of the common duct has been condemned because of the trauma its removal is said to inflict. This criticism is not justified in the experience of many surgeons, but they are careful to select T tubes of especially soft and flexible rubber. The Robson hepaticus tube is a safe, and, at the same time, very efficient means for draining bile to the outside.

The operation of cholecystectomy demands accurate technic and thorough knowledge of the regional anatomy. The procedure [7] insures comparative safety even in the presence of anomalous ducts and blood vessels. However, lesions may so alter the relations of structures that accuracy and definition become extremely difficult. Empyema and gangrene of the gallbladder, or stones impacted in the cystic duct are conditions that particularly predispose to injury because of the distortion at the juncture of the cystic and hepatic ducts with the common duct. On the other hand, injury may follow a simple cholecystectomy. Balfour [8] and McArthur [9] have had this experience. Fortunately, in both instances, the injury was immediately recognized and repaired, with complete and ultimate recovery of the patients. Stricture is the most serious result of injury, and may develop months or years later. Practically all reported cases of stricture have followed operations for disease of the gallbladder or bile ducts.

Stricture should be regarded as one of the unfortunate consequences of long delay in treatment, if there are symptoms of disease of the biliary tract. That stricture can occur without obvious cause is shown by the case reported by Phillips.[10] A man, aged 60, presented himself because of obstructive jaundice. He died from exhaustion five months later. There was no history of disease of the biliary tract. Necropsy revealed a stricture of the common hepatic duct which resembled cartilaginous stricture of the urethra. Only the outer coats of the duct were involved, the mucous surface being normal.

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7. Judd, E. S.: Cholecystitis; Changes Produced by the Removal of the Gallbladder, Boston M. & S. J. **174**:815-825 (June 8) 1916.

8. Balfour, D. C.: The Technic of Hepaticoduodenostomy, with Some Notes on Reconstructive Surgery of the Biliary Ducts, Ann. Surg. **73**:343-347 (March) 1921.

9. McArthur, L. L.: Repair of the Common Bile Duct, Ann. Surg. **78**: 129-139 (Aug.) 1923.

10. Phillips, Sidney: Simple Stricture of the Bile Duct: Peritoneal Surgery for Acute Suppuration and for Tubercular Disease, Lancet **2**:916-918, 1887.

Complete or partial obstruction to the flow of bile results in stasis in the liver, and the sclerogenic action of the stagnant bile produces biliary cirrhosis with serious impairment of hepatic function. Also, cholangeitis and extrahepatic infection often complicate the condition.

The liver possesses an enormous factor of safety. Experimentally, it has been shown that three fourths of the dog's liver can be obstructed without any clinical evidence of pigment or cholate accumulation, and nine-tenths can be obstructed without causing icterus of the tissues.[11] Normal hepatic tissue can undergo functional hypertrophy, and damaged tissue can regenerate. The clinical conditions that produce obstructive jaundice usually affect the entire liver, but, in exceptional cases, there may be a lesion of only one branch of the hepatic duct. Bristowe[12] gives a postmortem report of a man, aged 39, who had been jaundiced. There were cirrhosis and abscess of the liver and dilatation of the intrahepatic ducts, also a stricture, 0.64 cm. long, in the left hepatic duct. The right hepatic duct was healthy.

TYPE OF STRICTURE

Strictures of the bile ducts are either diffuse or partial. The type can be determined only at operation.

Diffuse stricture rarely comes to the attention of the surgeon. All the extrahepatic ducts may be involved in the cicatricial process. The condition occurs usually as congenital atresia of the bile ducts, and is most often due to obliterative cholangeitis.

Partial stricture is the most common, and may affect a large part of the common, rarely the hepatic, duct. Sometimes the common duct is represented by a small fibrous cord without a lumen. The usual condition is a definitely limited constriction of the duct, about 0.64 to 1.27 cm. long. Mathieu says that the stricture is rarely annular, but in our cases it has been typically annular and tubular. We have not made routine microscopic studies of the strictures in our cases. Competent observers report that the fibrous process affects chiefly the submucous layer of the duct. Instances, such as the case of Bristowe, occur in which the lumen of a stricture is lined with normal mucous membrane. Horsley,[13] in his experimental reconstruction of the common duct from a piece of jugular vein turned inside out, observed that normal epithelium may grow in and line the lumen of the new canal,

11. McMaster, P. D., and Rous, Peyton: The Biliary Obstruction Required to Produce Jaundice, J. Exper. Med. **33**:731-750 (June) 1921.

12. Bristowe: Cirrhosis; Abscess of Liver; Stricture and Dilatation of Ducts; Fatty Kidneys, Tr. Path. Soc. London **9**:222-223, 1858.

13. Horsley, J. S.: Reconstruction of the Common Bile Duct, J. A. M. A. **71**:1188-1194 (Oct. 12) 1918.

but that contraction and stenosis eventually occur. It is probable, therefore, that lesions involving only the epithelial layer of the ducts, whether of traumatic or infectious origin, may undergo complete repair by regeneration of epithelium. It does not seem reasonable that fibrosis involving only a part of the circumference of the duct could cause total obliteration of the lumen.

A duodenal ulcer near the papilla occasionally gives rise to biliary obstruction, and Moynihan [14] and others report cases of obstruction of the common duct above the papilla caused by cicatrization of a duodenal ulcer. These, however, are not true strictures of the common duct.

BENIGN STRICTURE OF THE COMMON DUCT

Forty-eight patients, thirty-seven women and eleven men, with benign stricture of the common duct, have been operated on at the Mayo Clinic. The youngest patient was 22 years, and the oldest 60. The incidence of stricture according to age was as follows: From

TABLE 1.—*Conditions Found at First Operation*

	Cases
Stones only in gallbladder	20
Stones only in common duct	3
Stones only in cystic duct	2
Stones in gallbladder and cystic duct	1
Stones in gallbladder, cystic duct and common duct	1
Stones in gallbladder and common duct	4
Empyema of gallbladder	5
Stricture of common duct	5
Cholecystitis only	13

20 to 30 years, five cases; from 30 to 40 years, fifteen cases; from 40 to 50 years, fifteen cases; from 50 to 60 years, eleven cases, and from 60 to 70 years, two cases. The average age was 41.4 years.

Condition at First Operation on Biliary Tract.—Lithiasis occurred in thirty-one cases, stones being found in the gallbladder, in the ducts, or in both. Cholecystitis without stones was present in fifteen cases. Five patients had empyema of the gallbladder, and eight had stones in the common duct. There were five patients in whom a stricture of the common duct was encountered at the first operation; two had stones in the common duct; one, empyema of the gallbladder, and two, only an associated cholecystitis. These are of interest because stricture is rarely seen at the first operation on the biliary tract (Table 1). No doubt more strictures of this type occur but do not become manifest until after operation, when a biliary fistula develops which shows no tendency to close, or when a decrease of jaundice is not evident after operation (Table 2).

14. Moynihan, B. G. A.: Duodenal Ulcer, Ed. 2, Philadelphia, W. B. Saunders Company, 1912, p. 282.

The common duct was opened and drained in ten cases, in eight of which a stone was removed.

The five cases in which a stricture was found at the first operation will be discussed under the treatment of stricture.

One case illustrates the sometimes almost infinite capacity of the human organism to withstand surgical measures. The history is briefly as follows:

CASE 2.—A man, aged 34, had had a cholecystostomy for pus and stones in the gallbladder three and one-half years before coming to the clinic; ten days later, a second operation was performed for a secondary collection in the wound; three months later, more stones were removed from the gallbladder, and a stricture of the common duct noted; two months later, a catheter was inserted into the common duct for drainage; six months later, an attempt to make a cholecystenterostomy failed, and resulted in a fecal and biliary fistula; three subsequent attempts were made to close the fecal fistula, but failed. When the patient came to the clinic, he had a biliary and fecal fistula; he was markedly jaundiced, had chills and fever, and the stools were clay colored. At

TABLE 2.—*Operations Preceding the Operation for Stricture*

	Operations Performed		
	Total	Elsewhere	Clinic
Cholecystectomy	44	24	20
Primary	23	17	6
Following cholecystostomy	10		
Combined with choledochostomy	7		
Combined with repair of duct for stricture	5		
Choledochostomy for stone followed by cholecystoduodenostomy	1		
Cholecystostomy followed by choledochostomy	1		
Eight previous operations on biliary tract	1		

operation, the fecal fistula was closed and the common duct, at the site of stricture, reconstructed over a rubber tube. He returned one year later because of a biliary fistula. The stools contained bile. Operation disclosed a stricture at the distal end of the common duct. This was dilated and a rubber tube fastened in. Three years later, he reported that he had good health but occasionally was jaundiced. He died from pneumonia seven years after the last operation.

SYMPTOMS AND DIAGNOSIS

Symptoms of stricture are not characteristic. The jaundice due to obstruction of the common duct may have several causes, such as stone in the duct, pancreatitis, cancer of the head of the pancreas, and cancer of the ampulla of Vater. The same conditions may be the cause of jaundice after operation on the biliary tract. Given a case in which there is jaundice associated with symptoms of stone in the common duct, at operation the surgeon excises a diseased gallbladder and removes one round stone from the common duct, which, after careful exploration, is found to be free, and then a drainage tube is inserted. The patient convalesces uneventfully, but several weeks later, when the tube is removed, he becomes jaundiced, and the stools become white. There is

a strong possibility of a stricture of the common duct. The first patient on whom W. J. Mayo [15] performed a hepaticoduodenostomy had had, five months previously, a cholecystectomy and drainage of the common duct for multiple stones in the gallbladder and duct. Again, as in the case reported by Walton,[16] a cholecystectomy was performed for gangrenous cholecystitis and a stone removed from the cystic duct. A complete biliary fistula developed, and a later operation revealed a stricture of the common duct, probably resulting from pressure necrosis from the stone in the cystic duct.

Four patients in our series became deeply jaundiced a few days after operation on the biliary tract. Two had had cholecystectomy elsewhere. One had had cholecystostomy elsewhere, and severe hepatic colics began eight months later. At operation in the clinic, a densely adherent gallbladder was removed. Jaundice and white stools appeared five days later. The other patient came with a biliary fistula, alternating with jaundice, which followed an operation elsewhere for drainage of the gallbladder. At a later operation, these four patients were found to have a stricture of the common duct. There is little doubt that a stricture had rather definitely formed before the first operation.

Ten patients [17] did not develop symptoms of stricture until six months or more after operation on the biliary tract; the average interval was thirty-five and two-tenths months. One patient had had a cholecystostomy for stones elsewhere. Six years later he developed a biliary fistula, and at operation a stricture was found at the juncture of the cystic and hepatic ducts. In all probability, the stricture was the result of an ulcerating stone which had been passed. In five cases, a stricture of the common duct was noted at the first operation. In the remaining twenty-eight cases, the interval varied from one to twenty weeks, the average being eight weeks after the first operation on the biliary tract.

SUMMARY OF THE SYMPTOMS

Forty patients had a history of jaundice; it was constant from the time of onset in twelve, two of whom had a biliary fistula; and it was intermittent in twenty-eight, eight of whom had a biliary fistula.

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15. Mayo, W. J.: Some Remarks on Cases Involving Operative Loss of Continuity of the Common Bile Duct, Ann. Surg. **42**:90-96, 1905; Resection of the Bile Passage After Serious Injury to the Common or Hepatic Ducts, Surg., Gynec. & Obst. **22**:1-6 (Jan.) 1916.

16. Walton, A. J.: Reconstruction of the Common Bile Duct, Brit. J. Surg. **9**:169-178 (Oct.) 1921; Discussion on the Surgery of the Common and Hepatic Bile Ducts, Reconstruction of the Ducts, Proc. Roy. Soc. Med. (Sect. Surg.) **17**:24-32 (Nov.) 1923.

17. Judd, E. S., and Burden, V. G.: Postoperative Stricture of the Common Bile Duct, Ann. Surg. **80**:210-216, 1924.

Seven patients did not have jaundice but had a biliary fistula. Twenty-three patients had attacks of severe hepatic colic followed by jaundice which would later disappear. Twenty patients had a biliary fistula; in eleven, it was constant, and, in nine, intermittent.

In the group of patients having attacks of colic and jaundice that were sometimes associated with chills and fever a stone in the common duct was strongly suspected. In our study, nearly 50 per cent. of strictures of the common duct gave rise to symptoms of intermittent biliary obstruction.

LOCATION OF THE STRICTURE

The location of the stricture is of decided practical importance with regard to the etiology and treatment. The usual situation is at the juncture of the cystic and hepatic ducts. In twenty-seven of our forty-seven cases, the stricture involved this area. We believe this site is determined by three chief factors: (1) It is the most common point of injury to the ducts; (2) it is the part of the ducts most often involved by extension of inflammation from the gallbladder, or pressure from a stone in the cystic duct, and (3) this area is the least dilatable portion of the ducts. Stricture at this point has been observed even in the presence of a stump of cystic duct. Stricture lower in the supraduodenal portion of the duct occurred in seventeen cases. In this location, the stenosis was probably the result of ulcerating stone, choledochitis or involvement of the duct in severe infection of the wound. In two cases, there was a stricture at the papilla. This is a common site for congenital stricture because the natural lumen of the duct at this point is very small and, consequently, is seriously affected by a descending angiocholitis. The diaphragm type of stricture is common in this region, and the exploring instrument easily breaks through it.

There was one case of stricture of the common hepatic duct in our series. This location is extremely rare. Deaver [18] has observed one case. He dilated the stricture through an opening in the common duct. The patient has remained well for five years.

TREATMENT OF STRICTURE

Stricture of the bile ducts is preventable in many cases, but a certain number will always occur because of the nature and condition of the disease. We might call attention to three chief factors in the prevention of stricture: early recognition and early surgical treatment of diseases of the gallbladder and bile ducts, thorough and accurate technic in the surgical procedure, and a complete primary operation whenever possible. The statistical method for determining the efficiency of a certain opera-

18. Deaver, J. B., and Ashhurst, A. P. C.: Surgery of the Upper Abdomen, Ed. 2, Philadelphia, P. Blakiston's Son & Co., 1921, p. 507.

tion is of limited value, and, in the case of operations for stricture of the bile ducts, it holds a place of even less importance. W. J. Mayo [19] has said that there are three fundamental principles that affect the welfare of patients treated surgically. They concern: (1) mortality from the operation; (2) benefit from the operation, and (3) disability following the operation. In view of these principles, the patient with stricture of the bile ducts should be considered a serious surgical risk whose mortality rate is high, but whose prospects of relief or cure without surgery are even less than in malignant disease. Such patients are usually deeply jaundiced and in an extremely weakened condition, as a result of impaired hepatic function. Regarding the operation itself, Moynihan [20] says:

> Secondary operations upon the biliary system are among the most difficult tasks in surgery. To obtain the best results all the highest qualities of the surgeon may be called into play: courage, resource, patience, accuracy and rapidity of judgment, the finest craftsmanship, and a tranquillity of mind and action that nothing can disturb.

The five cases in which a stricture was encountered at the first operation will be considered separately.

Case 3.—The gallbladder was diseased, and a considerable portion of the common duct strictured. Cholecystectomy was performed, and the hepatic duct united to the duodenum over a piece of rubber tube. The patient has now been perfectly well for seven and one-half years.

Case 4.—There was empyema of the gallbladder, a stone in the cystic duct, and stricture of the common duct. The gallbladder was removed, and the stricture excised. The divided ends of the common duct were then united posteriorly and left open anteriorly for drainage. The patient is perfectly well, nine years after operation.

Case 5.—There was cholecystitis and a stricture at the juncture of the cystic and common ducts. The gallbladder and strictured portion of the duct were excised and immediate end-to-end suture of the duct made. The patient had a recurrence of trouble, seven months later, and at a second operation the stricture, which had re-formed, was incised and the duct reconstructed over a T tube. The patient has remained perfectly well now for eight years.

Case 6.—Cholecystitis, stone in the common duct and stricture of the common duct were present. Cholecystectomy was performed, and the stone removed from the common duct. The stricture was excised, and the hepatic duct united to the common duct over a T tube. The patient has remained well for two years.

Case 7.—The diagnosis was cholecystitis, stone in the common duct, and stricture of the common duct. Cholecystectomy was performed, the stricture incised, and the duct reconstructed over a T tube. The patient left the hospital apparently well, but has not been heard from.

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19. Mayo, W. J.: An Address on the Surgery of the Hepatic and Common Bile Ducts, Lancet **1**:1299-1302 (June 30) 1923.

20. Moynihan, B.: The Recurrence of Symptoms After Operation on the Common Duct, Proc. Roy. Soc. Med. (Sect. Surg.) **17**:8-14 (Nov.) 1923.

These results indicate that primary operations for stricture are highly successful.

SUMMARY OF OPERATIONS AND RESULTS OF TREATMENT

A simple incision of the stricture was made in two cases. One of the patients was not relieved, and one died four and one-half years later. The other had a recurrence, two months later. The stricture was then resected, and end-to-end suture of the duct made. There was again a recurrence, and the stricture was again resected, and an anastomosis made over a T tube. Symptoms recurred, and finally the hepatic duct was united to the duodenum over a rubber tube. The patient was apparently well, on dismissal from the hospital, but has not been heard from.

Incision of the stricture and insertion of a T tube were made in four cases. One of the patients died in the hospital; one is well, five and one-half years later. One had a recurrence, six months later, and the side of the dilated duct was joined to the duodenum over a tube. The tube was vomited fifteen months after operation. The patient is living, three years later, but occasionally has an attack of jaundice. The other patient had a recurrence, six months later. The hepatic duct was then united to the duodenum over a rubber tube. The patient is living one and one-half years later, but becomes jaundiced at times. Roentgen-ray examination shows the tube still in place.

Incision of the stricture and insertion of a Robson drain were made in two cases. One patient had a recurrence after one and one-half years. The stricture was again incised, and a T tube inserted. The patient is living and well, seven and one-half years later. The other had a recurrence at the end of two months. The stricture was incised, and the duct reconstructed over a T tube. Again there was recurrence, and a plastic operation on the duct was performed over a rubber tube. The patient has not been heard from since his dismissal from the hospital.

Resection of the stricture and end-to-end suture over a rubber tube were made in two cases. One patient had a recurrence of the stricture at the site of anastomosis, two months later. Hepaticoduodenostomy was then performed. The patient is living, six years later, but occasionally has had an attack of colic and jaundice. The other passed the tube, five months after operation, and then had a recurrence of symptoms. Hepaticoduodenostomy was performed over a tube. The patient is living, one year later, but at times has colic and jaundice.

Resection of the stricture and end-to-end suture without a tube were made in three cases. One of the patients died fourteen months after operation. One is living and well four and one-half years later. The

other had a recurrence after nine months, and a reconstruction of the duct was made over a T tube. The patient is living and well, eight years later.

Resection of the stricture and end-to-end suture over a T tube were made in four cases. Two of the patients are living and well, two years later. One died in the hospital, and the other died, ten months after operation.

Hepaticoduodenostomy was performed in thirteen cases: over a tube in eight cases, and without a tube in five cases. Two of the eight patients are living and well, seven years later. One was unrelieved and died, six years later. Another was relieved, but died from other causes, two and one-half years later. One died in the hospital. One is living, three and one-fourth years later, but has colic and jaundice at times. One had a recurrence, after four months, then a second hepaticoduodenostomy was performed. The patient is living, one and one-half years later, but occasionally has colic and jaundice. One had a recurrence after seven months. A second hepaticoduodenostomy was then performed, and the patient is well, seven months later.

Four of the five patients who underwent hepaticoduodenostomy without insertion of a tube are living and well: one and one-half, two, two and one-half, and twelve and three-fourths years later, respectively. The other was not heard from.

Anastomosis of the common duct to the duodenum over a tube was performed in one case, and the patient is living and well, five and one-half months later.

Lateral anastomosis of the common duct to the duodenum was performed in one case, but was not successful. The patient died in the hospital.

Anastomosis of the tract of the external biliary fistula to the duodenum was performed in one case. There was a recurrence of symptoms after two weeks, and a hepaticoduodenostomy was performed. The patient was apparently well on dismissal, but has not been heard from since.

A plastic operation to enlarge the duct at the site of the stricture was performed in seven cases. One of the patients died in the hospital. Another was unrelieved, and died one year later. One was not heard from. One had thirteen operations on the biliary tract. One is living and well, two years later. One is living and well, six and one-half years later. One, who has lived two years after operation, has occasional jaundice.

The stricture was resected, the posterior half sutured, and the anterior half left open for drainage, in one case. The patient is living and well nine years later.

Reconstruction was made over a T tube after dividing the stricture in six cases. Two of the patients were unrelieved; one died four months, and the other, one and three-fourths years later. One had a recurrence in five months; then a resection of the stricture and an end-to-end suture were made. The patient is well, four and one-half years later. One had occasional jaundice two and one-half years later. One had a recurrence after two months; then a hepaticoduodenostomy was made over a tube. There was a second recurrence, after four months; another hepaticoduodenostomy was made without a tube. There was a third recurrence, after two months; then a third hepaticoduodenostomy was made over a tube. Nothing has been heard from the patient since his dismissal from the hospital. One was not heard from.

Anastomosis of the common duct to the duodenum without a tube was performed in one case, and the patient is well two and three-fourths years later.

Secondary operations because of recurrence of stricture were required in fourteen cases. Seven of the patients were treated by hepaticoduodenostomy. Of the fourteen patients, nine are living, one is dead, and four have not been heard from (Table 3).

TABLE 3.—*Summary of Results of Operations for Stricture*

Results	Number of Patients
Living	27
Perfectly well	19
Occasional colic or attack of jaundice	8
Died in the hospital	5
Died later	9
Not heard from	7

Two important factors enter into the treatment of stricture of the bile ducts: the patients are usually deeply jaundiced and immediate drainage of bile is imperative; free passage of bile to the intestine must be established before cure is complete. Perhaps the greatest task, in these operations, is the identification of the common duct. In a jaundiced patient without a biliary fistula, the best procedure is probably incision of the stricture and insertion of a Robson drain or a T tube to conduct the bile to the outside. If a cure does not result, a second operation to reestablish the bile channel can be more safely undertaken. There is often an associated cholangeitis in these cases, which improves after prolonged drainage of bile. If patients have a total biliary fistula, it is not advisable to attempt a reconstructive operation on the ducts until all signs of infection in the liver and ducts have subsided. The mere incision of a stricture and insertion of a drainage tube cannot be looked on as a curative measure because the stricture usually re-forms and symptoms return. There is no method corresponding to the passage of

sounds in urethral stricture to maintain the patency of the common duct after a stricture has been incised, and the flow of bile is easily obstructed because of the low secretory pressure of the liver.

Cholecystenterostomy is not practical for several reasons; the gallbladder has usually been previously removed; if present, it is diseased and not suitable for anastomosis, and the cystic duct is often obstructed, but chiefly because more than 50 per cent. of strictures occur at the juncture of the cystic and common ducts.

Plastic enlargement of the duct is usually impossible and rarely satisfactory.

From the experimental standpoint, resection of the stricture with immediate end-to-end anastomosis is an ideal method, and when it can be satisfactorily done is highly successful. Riggs [21] reports a case in which he resected the stricture and united the cut ends of the duct after the method of Carrel for blood vessel suture. The patient was perfectly well, three years later. There is a tendency, however, for a stricture to form at the site of anastomosis. The stricture was resected and some form of immediate anastomosis carried out in ten of our cases. Four patients were permanently cured; three had a recurrence of symptoms, and three died.

Attempts to form a new common duct from pieces of fascia. omentum, and from other tissues are doomed to failure because the new channel eventually contracts. It has been shown experimentally, by Horsley and by Sullivan, that new epithelium will grow along and line the new channel, but in the absence of a normal submucous layer, a progressive fibrosis and stenosis takes place. Results have demonstrated that the method of hepaticoduodenostomy, first developed by W. J. Mayo in 1905, is the most practical, most widely applicable, and most successful procedure for reestablishing the natural course of the bile flow. Attention is directed to the mistake of considering patients as cured when only a few months have elapsed since the operation. We have noted recurrence of symptoms after from two to four years of apparently complete relief. It is quite commonly observed in patients who have had operations for stricture that symptoms of intermittent biliary obstruction, such as colic and jaundice, may continue at infrequent intervals for from one to four years, and then entirely disappear. It frequently occurs that patients who have had a reconstruction of the common duct made over a rubber tube will remain comparatively free from symptoms as long as the tube is in position, but when it passes into the bowel or is vomited, symptoms of biliary obstruction promptly return. The presence of the tube can be demonstrated by the roentgen ray.

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21. Riggs, T. F.: End to End Suture of the Bile Ducts, Surg., Gynec. & Obst. **22**:660 (June) 1916.

TWENTY-SEVENTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY*

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CONGENITAL CONDITIONS

Origin of Congenital Dislocation of the Hip.—The importance of the upright position that man has assumed is pointed out by Krukenberg [1] as a fundamental difference between man and the lower animals. In the upright position the hips are developed in a position of extension, which position is a critical one from the standpoint of dislocation as the flexors of the hip are developed in a position of tension. Krukenberg has experimented on three dogs soon after birth, placing their limbs in the extended position that is assumed by man standing upright. In both female animals the hips were dislocated, while the male animal showed no change.

Spontaneous Healing of Congenital Dislocation of Hip.—Kopits [2] reports three cases of spontaneous reduction of congenital dislocation of the hip and believes that they occur much more frequently than is generally believed. He realizes the danger of proclaiming this fact because of its effect on parents who might hesitate to subject their children to proper treatment. From his studies of literature and from his own cases, he was led to believe that spontaneous reduction may take place only in early childhood. After the second year spontaneous reduction may not be expected. Also, spontaneous reduction will take place only when the head is in the immediate neighborhood of the acetabulum. He believes from this that it is justifiable to wait until the end of the second year before making an attempt to reduce a congenital dislocation.

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*This report of progress is based on a review of 258 titles selected from 452 titles dealing with orthopedic surgery, appearing in medical literature between Dec. 6, 1924, and April 1, 1925. Only those papers which seem to represent progress have been selected for note and comment.

1. Krukenberg: Zentralbl. f. Chir. **52**:304 (Feb. 7) 1925.

2. Kopits: Ztschr. f. orthop. Chir. **46**:158, 1925.

Fate of Unreduced Congenital Dislocation of the Hip and the Bifurcation Method.—After discussing the problem of the unreduced dislocation of the hip, Lorenz [3] believes that congenital dislocation is not, in this sense, a true dislocation but that it is due to physiologic changes in the structure of the pelvis and the hip joint. He points out the fact that the female sex is much more frequently affected than the male, and finds explanation for this in the slight forward torsion of the acetabulum in the female, as pointed out by Graf and Fehling. In such subjects as have a predisposition to dislocation without actual displacement, there is apt to develop an arthritis deformans of the hip, as pointed out by Calot; also, coxa vara or osteochondritis may appear in these hips.

He describes the process of dislocation as follows: The upper edge of the acetabulum is flattened by the head of the femur, and a furrow results leading to the initial displacement. The forward twist of the neck of the femur may then increase. In certain cases spontaneous reduction may result from prolonged rest in bed due to illness, but unfortunately this spontaneous reduction is not always lasting. The old unreduced case presents a femoral head that wanders further and further, and finally the pelvis hangs on the elongated capsule of the hip joint; the head of the femur has gone far backward and upward and the acetabulum is nothing but a small triangular depression.

The so-called bifurcation operation that he recommends has for its object the dislocation of the lower fragment and the fixation of the fragment in the acetabulum. He had done this operation in more than 100 cases and believes that the results are encouraging and the operation harmless compared with the difficult, shock-producing operations that attempt actual reduction. He believes that there is a wide use for this bifurcation operation in other conditions than congenital dislocation, such as pathologic dislocation following infection, trauma, or dislocations due to paralysis, when replacement is impossible, and also in cases of severe coxa vara. He recommends it further in cases of nonunion following fractured neck of the femur, painful arthritis deformans, and wandering acetabulum in old tuberculosis of the hip joint. Lorenz also believes that the operation has a place in acute hip disease and recommends its consideration in cases in which there is rapid destruction of the hip joint.

[Ed. Note.—We feel that this so-called bifurcation operation of Lorenz is not to be seriously considered as an advisable surgical procedure, except perhaps in rare instances. We do not agree in the widespread use of the operation that he recommends. We feel that

3. Lorenz: Ztschr. f. orthop. Chir. **46**:124, 1925.

the operation is mechanically unsound and that it represents a non-surgical attitude, in that its main advantage seems that it is a "short cut" method of treatment.]

Congenital Subluxation in Pathogenesis of Hip Disease.—Calot [4] has made an anatomic study of 700 hips at necropsy and is convinced that one out of every thirty persons, regardless of age, shows some trace of a congenital subluxation of the hip joint. Partial dislocations are about ten times more frequent than are complete dislocations. The congenital anomaly known as congenital dislocation represents a local lack of resistance in the hip. He believes that 75 per cent. of the cases diagnosed as tuberculosis of the neck of the femur, as well as 50 per cent. of the cases that are called coxalgia may be due to congenital malformation of the hip. In the necropsies of eighty cases of rheumatoid arthritis, the hip joints showed that 96 per cent. had congenital subluxation. He believes that all cases of Legg's disease are nothing but congenital luxations of the hip joint. Roentgenography has shown in certain of these cases spots in the epiphysis and in the neck of the femur which he believes are often mistaken for tuberculous foci. These spots are due to defective nutrition from disturbed calcium metabolism.

[Ed. Note.—We believe that radical and sweeping as may be these assertions, Calot has presented a point of view that should be seriously considered in the possible explanation of some of the growth disturbances that occur in the hip joint.]

Operative Correction of Equinovarus.—Straus [5] points out that persistent clubfoot with calcaneus varus demands the following operative procedure for correction: The major attack should be made on the os calcis. This bone should be divided by a chisel cut from the inner side toward the outer side, not entirely through, but only so far as the deformity of the os calcis may be corrected. The gap created by correcting the deformity should be filled in with pieces of bone taken from the os calcis. The Achilles tendon should be lengthened and the tendon of the posterior tibial muscles cut.

First Dorsal Rib Simulating Cervical Rib.—Brickner and Milch [6] report two types of cases: one, in which there is obvious deformity of the first rib, presents symptoms of nerve pressure, and a second, which has no apparent rib deformity, gives exactly the same symptoms. They quote from the literature many explanations and believe that Todd's is the most rational. Todd believes that in the development of the arm

4. Calot: Bull. de l'Acad. de méd. **93**:77 (Jan. 13) 1925.

5. Straus: Zentralbl. f. Chir. **52**:301 (Feb. 7) 1925.

6. Brickner, W. M., and Milch, H.: Surg., Gynec. & Obst. **40**:38 (Jan.) 1925.

bud, the axis of growth is at the level of the seventh cervical segment, and consequently that the seventh cervical root should be the central and largest nerve root in the arm appendage. The eighth and ninth nerve roots curve upward to the level of the seventh root and then downward to their distribution in the arm. Any influence that may cause tension of the eighth or ninth roots that form the inner cord of the brachial plexus would give rise to the symptoms observed in the cervical rib or in a first dorsal simulating a cervical rib. An elevation of the rib relative to the shoulder girdle, or a depression of the shoulder relative to the rib, will satisfy the conditions necessary for the appearance of symptoms. This explanation makes the whole syndrome quite clear. The local signs recede in importance and become merely incidental findings. The vascular symptoms are to be regarded in the light of effects produced by traction either in the sympathetic fibers contained in the first dorsal root or on the subclavian artery itself pressed against the rib. The nerve symptoms appear to be the result of traction rather than a transverse compression. With trauma there is excitation of the neurologic symptoms which cause the loss of tone of the trapezius or the downward displacement of the shoulder, and would lead to a stretching of the nerve. Brickner and Milch believe that the treatment is obviously nonoperative or operative. All patients should be given a trial of methods of support and muscle training. In cases in which there is continued pain or progressive nerve or vascular interference, if no relief is seen by conservative measures, resection of the rib should not be delayed. They believe that the operative procedure has been well described by Stiles and by Telford in Stopford's article.

TUBERCULOSIS

Joint Tuberculosis.—Smith [7] reports the results of a large series of Pirquet tests and inoculation tests in cases in which the diagnosis of tuberculosis of the bones and joints was confirmed by operation and pathologic examination. The only laboratory test that establishes the diagnosis is guinea-pig inoculation, and this may be inconclusive or negative. The condition is obscured in many cases by immobilization. The percentage of error in diagnosis in tuberculosis of the bones and joints is high. The mistake of calling a nontuberculous joint condition tuberculosis probably is just as frequent as the reverse. As a result of error in diagnosis, much harm is done to patients, and the statistics on the result of conservative treatment are very misleading. Because of the importance of being certain of the diagnosis, exploratory opera-

7. Smith, A. D.: Early Diagnosis of Joint Tuberculosis, J. A. M. A. **83**: 1569 (Nov. 15) 1924.

tion is indicated in all doubtful cases. When a positive diagnosis has been made without proper verification, and treatment by immobilization has been carried out for a longer or shorter period, diagnosis is made much more difficult from clinical observation.

[ED. NOTE.—The importance of Smith's observations are not generally realized. It is the general custom to make positive diagnosis of tuberculosis of the bones and joints without proper confirmatory tests. We feel that in our own experience many serious errors and much prolonged and unnecessary treatment has been prevented by the methods of diagnosis that are reaffirmed by Smith's article.]

Rib Graft in Pott's Disease.—Gutierrez [8] prefers to use a rib implant in Pott's disease or injuries of the spine rather than a graft from the tibia. He believes that on account of the structure, its length and its flexibility, it is a better type of graft, and again that it is easily taken because of its proximity to the spine. He reports exceptionally satisfactory results in six patients.

POLIOMYELITIS

Specific Precipitin Reaction in Epidemic Poliomyelitis.—Rosenow [9] has studied a large number of epidemic and sporadic cases of poliomyelitis and offers a test that he hopes will prove of value. The test is described as a specific precipitation, resulting from a union between precipitogen in the cleared extract and precipitin contained in the serum. As a result of the precipitin reaction done on animals, it seems warrantable to conclude that infection of the upper respiratory tract occurs constantly in patients with acute poliomyelitis and that during epidemics a large number of normal persons have this respiratory tract infection. Perhaps disappearance of epidemics and freedom from poliomyelitis for some years after an epidemic are due in part to immunity acquired through infection by this streptococcus, even without invasion of the central nervous system. In any event, a positive reaction seems not without significance because within a week after the throat swabbings proved positive, two persons are known to have developed severe attacks of poliomyelitis and three had abortive attacks. The question whether a positive reaction indicates the presence of the virus in addition to the streptococcus is still uncertain and is at present under investigation. Since the reaction is positive at the onset of poliomyelitis even before paralysis has developed, the test should find its greatest usefulness as an aid in early diagnosis and hence in early serum treatment. Since the reaction becomes negative in most cases in from two

8. Gutierrez, Alberto: Arch. franco-belges de Chir. **27**:768 (Sept.) 1924.

9. Rosenow, E. C.: Specific Percipitin Reaction in Epidemic Encephalitis, J. A. M. A. **84**:429 (Feb. 7) 1925.

to three weeks, it may prove a simple means of determining when quarantine may be lifted with safety. Moreover, it may also prove of value in epidemiologic studies of poliomyelitis.

Study of the 1923 Epidemic of Anterior Poliomyelitis in Kansas; Preliminary Report.—Dively [10] believes that if an early diagnosis is made and proper treatment instituted during the febrile stage, the resulting cord involvement is not only lessened, but in a great percentage of cases prevented. If proper treatment is carried on during the second or convalescent stage, from 75 to 85 per cent. of the cases will show marked improvement or complete recovery, against from 40 to 45 per cent. of partial recovery if proper treatment is not carried out. If proper treatment is carried out during the first and second stages, fully 90 per cent. of the deformed limbs and backs can be prevented. Many patients recover spontaneously without treatment, while others still show signs of paralysis after one year of careful supervision. There are definite anterior poliomyelitis carriers. The infection is selective and may run through neighborhoods or families, picking out various individuals. Doubtless, most persons in a community during an epidemic have the disease in the right or abortive form, which gives immunity.

Lumbar Puncture in Spinal Paralysis.—Wolf,[11] discussing the recent paper of Schanz on lumbar puncture in paralysis, states that he has used that method for many years. He is not able to discover in literature accurate data on the findings of this procedure. In his own cases, he found that the pressure of the spinal fluid was much increased and that the amount of albumin was much increased. He concludes that repeated lumbar puncture is indicated in spinal paralysis, as it may relieve edema and swelling of the inflamed spinal cord. He has performed spinal puncture in forty-three cases with but one death in a case of very grave and furious onset. In almost all cases improvement was rapid and extensive beyond expectation. In several cases, all signs of paralysis disappeared within a few days. He recommends the repetition of punctures until normal conditions are found in the spinal fluid.

RICKETS

Ultraviolet Rays in Treatment of Rickets.—Lesne and de Gennes [12] consider the ultraviolet rays as an excellent treatment of rickets in young children. The effect is not only local but general. Their theory is that the rays, by way of the sympathetic system, exert an action on the

10. Dively, R. L.: J. A. M. A. **84**:85 (Jan. 10) 1925.

11. Wolf, S.: München. med. Wchnschr. **72**:56 (Jan. 9) 1925.

12. Lesne and de Gennes: Paris méd., Dec. 20, 1924, p. 520.

endocrine glands. This modifies favorably the calcium and phosphorus metabolism and the assimilation of phosphorus.

Experiments on Depriving Living Bones of Lima Salts.—Strauch,[13] in ten cases of rickets occurring at the age of from 2½ to 4 years, with marked deformities of the legs, has suggested the possibility of softening the bones by venous congestion for twenty hours, and by the administration of ammonium chlorid in small doses. In this way the bones, which are hard, became soft and it is possible to correct the deformity and place the limbs in plaster-of-Paris and allow the bones to reharden. His observation is based on animal experimentation. Strauch suggests that other measures that are known to create rickets might be used as aids in softening bones, such as depriving the patient of sunlight and withholding vitamins during the period of softening.

Frequency of Rickets.—Maurer,[14] in Munich at the Children's Hospital, has gathered statistics on the examination of 4,000 children. Of these, in the first and second year 70 per cent. showed signs of rickets. At the twelfth year, 24 per cent. showed signs of rickets. This means that about one third of all rachitic children have lasting damage done by the disease. The frequency of rachitic disease was relatively large among children born in the years of the war; that is, from 1914 to 1917. Maurer believes from his study of pedigrees that there is an hereditary frequency in rickets itself, and also in the similarity and anatomic location of the deformities of rickets.

Climatologic Observation.—Forbes and Green,[15] from their study of the relation of the prevalence of rickets and the amount of sunshine in Colorado, draw the following conclusions: 1. Severe rickets is not common in Colorado, as evidenced by the relatively few orthopedic corrections necessary. Only one case of bow-legs was encountered in a survey of 503 infants. 2. Mild rickets is much less common in Colorado than in the eastern states. 3. Rickets in Colorado reaches its highest incidence in the latter half of the first year and is relatively uncommon in the second year, at the age when rachitic deformities are frequently noted. 4. Beading of the ribs was the commonest physical sign, and the evidence of this sign bears a close relation to the incidence of rickets as diagnosed in a clinical survey. 5. Epiphyseal enlargement was the second most frequent sign noted. 6. Craniotabes was found to be associated with other signs of rickets in 9.4 per cent. of the babies from 4 to 8 months of age, and with no signs of rickets in 5.8 per cent. of this age group. 7. Head sweating, although considered an unreliable sign, was commonly associated with other signs of rickets, especially

13. Strauch, C. B.: Arch. f. klin. Chir. **134**:181, 1925.
14. Maurer: München. med. Wchnschr. **72**:286 (Feb. 13) 1925.
15. Forbes, Roy, and Green, Berryman: Colorado Med. February, 1925, p. 69.

beading. Head sweating without any signs of rickets was present in from 3.8 per cent. to 7.9 per cent. of the cases in all age groups. 8. The highest incidence (78.2 per cent.) of rickets and the most marked cases were encountered in the small group of colored babies examined; the lowest incidence (14.73 per cent.) was found in an Italian welfare station. 9. Denver receives from 18 to 21 per cent. more sunshine during the winter months than either New York, Chicago or New Orleans. 10. The ultraviolet light intensity is greater at all seasons of the year in Denver than in the other cities considered. 11. The low incidence of rickets may therefore be attributed to the high percentage of sunshine afforded during the winter months and the intensity of the utraviolet light content at this altitude.

OSTEOMYELITIS

Brodie's Abscess.—In the thirteen cases cited by Henderson and Simon,[16] the staphylococcus was found in three cases; examination for organisms was negative in five cases, and no such examination was recorded in five cases. In five cases a special search was made by microscopic examination and animal inoculation for evidence of tuberculosis, with negative results. Evidence of pulmonary tuberculosis was found in only one case, in which inactive tuberculosis of both upper lobes was demonstrated by the roentgen ray. Clinically, the chest findings were negative, and guinea-pigs inoculated with the material from the abscess gave negative results. It is probable that the staphylococcus is the usual infection.

ARTHRITIS

Serologic Significance of Streptococci in Arthritis and Allied Conditions.—Burbank and Hadjopoulos,[17] under an improved and more painstaking technic, report very satisfactory results in the study of the serology of streptococcus infection, and are led to make the following conclusions: In using active serum they have developed a technic for bacterial fixations, in the main of the streptococcus types, whereby various arthritic and rheumatoid conditions may be classified serologically into three major groups: (1) Arthritis reacting to hemolytic streptococci and belonging to the iso-atrophic class. This type in pure form is periarticular. (2) Arthritis reacting similarly to hemolytic streptococci but of different fixing properties. This type is aniso-atrophic or deformans. (3) Arthritis reacting to streptococci of the *Streptococcus viridans* type and belonging to the ostearthritic or productive form.

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16. Henderson, M. S., and Simon, H. E.: Arch Surg. **9**:504 (Nov) 1924.

17. Burbank, Reginald; and Hadjopoulos, L. G.: J. A. M. A. **84**:637 (Feb. 28) 1925.

The bony change is often a very early manifestation in this group and is demonstrated by crepitus and by roentgen ray. The majority of arthritic cases that are not arrested or cured early in the course of the disease have a tendency to undergo further changes leading to mixed types. Thus, in an active exudative periarthritis or in an arthritis deformans, the bony parts gradually may become involved and give rise to a mixed serologic picture. Similarly, in a chronic productive arthritic case, acute tonsillitis or some other hemolytic infection may engraft a synovial (hemolytic) flareup on a chronic hypertrophic joint. A fair percentage of arthritic patients, especially those suffering from colitis and chronic constipation, have a marked tendency to effect complement fixation in certain strains of nonhemolytic cases. This type of organism seems to be a transition form of streptococcus hemolyticus, with certain properties lost through secondary intestinal implantation. Certain pathologic conditions have long been known clinically to be precursors of arthritis. Serologically, they have confirmed these clinical observations. In controlling the value of the test in normal cases, the possibility of the presence of some masked focus of infection must be considered. With this point in mind, the test is not diagnostic for arthritis alone, but is diagnostic of a wide group of acute and chronic infections that give rise to antistreptococcic bodies in human serums.

Arthritis of the Menopause; a Study of Fifty Cases.—From their study of cases Cecil and Archer [18] have concluded that the arthritis of the menopause is a definite clinical syndrome. It is a chronic polyarthritis of obese, middle-aged women, occurring at or just after the menopause, and is characterized by persistent stiffness and pain in the joints affected. It is noninfectious in orgin. This form of arthritis appears to be definitely related to the endocrines, but the exact nature of this relationship has not been established. The disease runs a mild chronic course.

Function of Joints and Arthritis Deformans.—Buckhardt [19] has done some experiments on animals designed to determine the relationship between the function of the joints and arthritis deformans. He has injected various irritating fluids into the joints, and in one series has allowed the animals free motion and in the other has employed fixation of the limbs. In the experiment in which free motion was allowed, the joint changes consisted of slight alterations of the cartilage up to complete degeneration of the same. In the experiment in which the joints were kept at rest, the joints remained unchanged for a while, but later showed complete destruction, ending in ankylosis. The author draws

18. Cecil, R. L., and Archer, B. H.: J. A. M. A. **84**:75 (Jan. 10) 1925.

19. Burckhardt, H.: Arch. f. klin. Chir. **132**:4,1924.

the conclusion that the regeneration is influenced by the mechanical and anatomic character of the joint.

NEOPLASMS

Modern Views on Etiology of Osteosarcoma.—Leriche and Policard [20] believe that the classic conception of osteosarcoma is not entirely correct, and should be changed in order to conform to newer views of osteogenesis. The osteoblast should not be considered as a specific element, but only as a variety of connective tissue cell. The osteoblast does not form the specific tumor. That is, an osteosarcoma is not an osteoblastoma. Leriche and Policard feel that osteosarcoma as well as osteochondrosarcoma is a common type of sarcomatous growth that originates from connective tissue of the bone. The osseous tissue contained in the tumor is not in any sense specific.

Errors in Roentgen Ray; Diagnosis of Osteogenic Sarcoma.—Beye [21] reports two cases of osteosarcoma which were apparently cured by roentgen-ray treatment, but which at necropsy showed no evidence of sarcoma whatever, being cases with proliferative changes in the bone due to pyogenic osteomyelitis and tuberculosis. Beye believes that the present diagnostic methods are not conclusive, as approximately 50 per cent. of error in diagnosis of supposed sarcomas of bone exists in the 454 cases submitted for registration. In some cases of osteogenic sarcoma, the roentgenogram is not distinctive. In others, the roentgenogram simulates that of pyogenic osteomyelitis so closely that a differential diagnosis between the two conditions is not possible from the plate alone. Beye feels that in late years, roentgen ray and radium have been used in an increasing number of cases diagnosed as sarcoma, and that satisfactory results have been reported. He feels that some of the cases that have been considered as cured are open to the question as to the correctness of the diagnosis.

Bone Tumors: Myxoma: Three Cases.—Bloodgood [22] points out that no surgeon should explore a bone tumor without being prepared to cauterize the wound. He believes that the actual cautery is far safer than the chemical cauteries, and also that the best cautery is the cheapest—the plumber's soldering iron. He calls attention to the fact that the only cases of myxoma or myxosarcoma that have been permanently cured have been subjected to amputation or resection without exploratory incision. This covers all cases except those of the phalanges and metacarpals, and a single cases of disease in the astragalus. Every myxoma

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20. Leriche, R., and Policard, A.: Presse méd. **32**:957 (Dec. 3) 1924.
21. Beye, H. L.: Ann. Surg. **80**:730 (Nov.) 1924.
22. Bloodgood, J. C.: Ann. Surg. **81**:99 (Jan.) 1925.

which has been explored has recurred locally, except the case of Dr. Rockey in which the soldering iron was used, and which has shown no recurrence after nine years.

Multiple Xanthoma of the Tendons.—McWhorter and Weeks [23] point out that xanthoma tuberosum multiplex and all forms of xanthoma are the result of a systemic disease in which hypercholesterolemia is an essential feature. The pathology of these growths probably represents an irritative connective reaction due to the deposit of cholesterin. Since recurrence of these growths probably follows their removal, surgical intervention is indicated only in those cases in which there is loss of function or pressure disturbance.

Fibrocystic Disease of Bone.—Muir [24] reports two cases of fibrocystic disease of the bone, both occurring in girls 14 years old, and both showing involvement of the femur, one at the upper end and one in the lower. From a study of the pathologic material it would seem that in these cases the preliminary change was the appearance of fibrous tissue in the marrow which became entirely replaced. Later, an invasion and absorption of the compact bone occurred. A still later development was the formation of cystic spaces within the fibrous tissue, the coalescence of these leading to larger cysts. Such a condition, if it extended, would inevitably lead to secondary fracture. The preliminary treatment in each case was the curetting away of all the affected tissue, and this, if done sufficiently early and thoroughly, would seem sufficient. Should the condition progress, however, amputation appears to be the only available treatment.

Primary Sarcoma of the Trochanteric Bursa.—Razzaboni [25] reports a case of primary sarcoma of the trochanteric bursa occurring in a woman of 62. The tumor was of about two years' duration. It was easily removed, was entirely encapsulated, and had no apparent connection with the trochanter. It is of interest chiefly from its great rarity.

GROWTH DISTURBANCES OF BONE

Contribution to the Pathology of Inflammation of the Tarsal Scaphoid in Children.—Lecène and Mouchet [26] have found only one case of tarsal scaphoid inflammation in the fifty or more cases reported in which there was histologic examination of the tissue. This case was that of Behm of Rostock, who concluded that the necrosis was due to

23. McWhorter, J. E., and Weeks, Carnes: Surg., Gynec. & Obst. **40**:199 (Feb.) 1925.

24. Muir, J. B. G.: Lancet **2**:1170 (Dec. 6) 1924.

25. Razzaboni, Giovanni: Chir. d. org. di movimento **9**:87 (Dec.) 1924.

26. Lecène, Paul; and Mouchet, Albert: J. de radiol. et d'électrol. **8**:357 (Aug.) 1924.

anemia. The pathology of this condition, therefore, is hypothetic. Koeler and Abramsan believe that it is an anomaly. Stumme and Schultze believe that it is due to a compression fracture. Mouchet and Roederer consider it is a dystrophy. The case here reported shows microscopically that the condition is one of a low grade osteomyelitis. The authors are inclined to apply this to one case only, and do not suggest that all cases are due to this cause.

Köhler's Disease of the Tarsal Scaphoid.—Kidner and Muro [27] believe that Köhler's disease is an interference with the power of bone production in the scaphoid of the foot. This interference is due to low grade infection, the source of which is unknown. This infection attacks the scaphoid for two reasons: because the scaphoid is the bone in the arch of the foot which bears the greatest strain, and because it has the poorest blood supply of any of the bones of the tarsus. Trauma undoubtedly has an influence in making the scaphoid susceptible to the infection. Clinically the symptoms and signs are all those of a low grade infection. In the previous pathologic examinations the picture has been that of infectious irritation. In their case, the picture is not compatible with infection. The infection is so mild that ordinarily it is overcome by the resistance of the body, and development goes on with an approach to the normal. Treatment of this condition is of the greatest importance, because it is necessary to protect the damaged nucleus from strain and crushing, in order that flatfoot or weak foot may be avoided in later life. The treatment should be continued rest. This rest should be in plaster-of-Paris with the foot in strong overcorrection in varus and cavus. It should be continued for from two to three months.

Discussion on Köhler's Disease: With a Report of a Case.—Hodgkinson and Duke [28] report a typical case in a 6 year old boy. They are inclined to believe that chronic infection may explain the condition, giving as their reason the fact that there is diminution in the growth of the bone and sclerosis of the same, which suggests chronic osteitis. They also believe that the condition may be due to an interference with osteogenesis in the navicular nucleus brought on by an infection that is too mild to produce a destruction, but which ends in osteosclerosis.

Etiology of Pseudocoxalgia. A Critical Survey of the Congenital Theory (With a Reply by Murk Jansen).—Perkins [29] has made an exhaustive study of the etiology of pseudocoxalgia. It is not sufficient to say that he reports Jansen's congenital theory that the thickened

27. Kidner, F. G., and Muro, Felipe: J. A. M. A. **83**:1650 (Nov. 22) 1924.

28. Hodgkinson, H. R., and Duke, L. S.: M. J. Australia **2**:552 (Nov. 22) 1924.

29. Perkins, George: J. Bone & Joint Surg. **7**:18 (Jan.) 1925.

acetabulum or the ischium varum with its shallow socket is the cause of the flattened head. He does not question the restriction of the amniotic sac as a cause of the congenital abnormality of the pelvis, but doubts that the primary deformity of a shallow hip socket results in the secondary anatomic changes in the head and neck of the femur. Jansen supports his contention in an able reply and the etiology is still in doubt.

Pseudocoxalgia.—Noble [30] draws his conclusions from a study of fifty-six cases in the Mayo Clinic. The congenital hypothesis of Jansen, the infectious theory of Legg and others, and the result of trauma are considered. No one of the foregoing hypotheses seems adequate to explain the etiology in all cases, and it seems highly probable that two of the factors may be involved, inherent congenital weakness at the epiphyseal line and trauma. The disease has much in common with adolescent coxa vara, although the age period and the position of the epiphysis, and consequently the deformities, are different. There appears to be a definite inherent weakness at the epiphyseal line, whereby trauma of a minor nature, not sufficient to cause displacement, but sufficient to cause vessel blockage to the epiphyseal line, may produce the subsequent changes.

A Localized Affection of the Spine Suggesting Osteochondritis of the Vertebral Body, with the Clinical Aspect of Pott's Disease.—Calvé [31] reports two cases of a hitherto undescribed pathologic condition of the vertebra closely resembling Köhler's disease of the scaphoid. It is similarly isolated in one bone of a group. In both of these cases tuberculosis and syphilis were ruled out. There was present a flattening of the body of a single vertebra, with much increased density and on either side a perfectly normal cartilage. Such an escape of the cartilage in tuberculosis cannot be conceived. Both cases had the usual symptoms of tuberculosis without abscess and were treated as such. There was no history of trauma nor evidence of infection. Undoubtedly this observation will bring to light other similar cases, as followed Köhler's report.

Osteochondritis of the Clavicle.—Friedrich [32] describes an alteration in the clavicle similar to that seen in osteochondritis of the hip. In the two cases that he reports there was painful swelling of the sternal end of the clavicle. A roentgenogram of the clavicle demonstrated spots in the end of the bone similar to those seen in Legg's disease. The two patients were operated on, and microscopic section of the tissue removed showed necrosis of the bone with no evidence of infection. He offers no explanation as to the cause of the disease.

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30. Noble, T. P.: J. Bone & Joint Surg. **7**:70 (Jan.) 1925.
31. Calvé, Jacques: J. Bone & Joint Surg. **7**:41 (Jan.) 1925.
32. Friedrich, H.: Deutsch. Ztschr. f. Chir. **187**:5-6, 1924.

Adolescent Coxa Vara.—From the standpoint of treatment, Noble [33] classifies patients with adolescent coxa vara in two groups: (1) those seen early before the bones have been allowed to fix in the deformed position, and (2) those seen late when bony union has been allowed to take place in the deformed position. Early diagnosis is important. All cases of slight injury to the hip during adolescence should be carefully roentgenographed, and a weight-bearing caliper applied if there is any suspicion of coxa vara.

Displacement of Upper Epiphysis of Femur Treated by Open Reduction.—Wilson [34] reports seven cases of "slipping epiphysis," five having a complete separation due to a mild trauma and accompanied by only a slight functional impairment and limping. There were four recent cases and three late ones in which malunion had taken place. The operation is practically the same in all cases. Through a broad incision (Smith-Petersen) the joint is exposed and the epiphysis freed and replaced and the hip put up in full abduction and internal rotation. The results have been excellent, giving a nearly normal hip. The patients were from 13 to 17 years of age, and all but one boys. Four were types showing definite infantilism and the other three of the tall, slender, rapidly growing type. It is believed that the standard of correction should be higher and that a closed reduction is often insufficient to attain this result.

A Separate Center of Ossification for the Tip of the Internal Malleolus.—Den Hoed [35] has examined 150 normal children by roentgen ray and has shown the presence of an "os tibiale" in twenty-one cases. In the cases in which this existed, the children were between the ages of 6 and 12 years. Fourteen of these cases were in boys; this differs from the findings of Fairbank, who reported that the condition was met only in girls. Den Hoed believes that there is a separate center of ossification at the tip of the internal malleolus and that the bone developed is not a sesamoid bone, as has been suggested by some writers.

BACK STRAIN

Sciatic Pain.—Danforth and Wilson [36] have made a study of the causes of sciatic pain, basing their conclusions on the dissection of twelve cadavers and the clinical findings in twenty-one patients. The anatomic studies showed that changes occurring at the lumbosacral junction have the greatest possibility of affected nerve structures. The

33. Noble, T. P.: Ann. Surg. **80**:773 (Nov.) 1924.

34. Wilson, P. D.: J. A. M. A. **83**:1749 (Nov. 29) 1924.

35. Den Hoed, D.: Brit. J. Radiol. **30**:67 (Feb.) 1925.

36. Danforth, M. S., and Wilson, P. D.: J. Bone & Joint Surg. **7**:109 (Jan.) 1925.

intervertebral canals between the fifth lumbar vertebra and the sacrum and between the fourth and fifth lumbar vertebra are the most likely points of nerve involvement. The solid nature of the sacrum offers little possibility for involvement of the sacral nerves during their exit, and the subsequent course of these nerves is such as to render their involvement unlikely. The clinical examination of the spine showed that in all these cases there was evidence of disturbance in the lower lumbar region. In the majority of these cases this was substantiated by positive roentgenologic findings in the same region. Clinical evidence of nerve involvement was found in fifteen out of the twenty-one cases, consisting of areas of anesthesia or hyperesthesia, changes in the reflexes, and points of tenderness over the nerve trunks. In many of the cases the signs were of a nature to aid in localizing the portion of the nerve affected and pointed to a lesion of the fourth or fifth lumbar nerve roots. The findings are reported in detail, with numerous charts and illustrations, and are deserving of detailed study.

[ED. NOTE.—Among orthopedic surgeons there are two schools of thought regarding sciatic pain of the type reported above. One regards it as a manifestation of disease or the sacro-iliac joints; the second considers it as secondary to some lesion affecting the lumbar spine and particularly the lumbosacral junction. The evidence compiled by Danforth and Wilson favors the latter point of view and is worthy of careful consideration.]

BONE AND JOINT SURGERY

Transplantation of Metatarsal Bones.—Rankin [37] reports four cases in which the second metatarsal, including the shaft and epiphysis, was transplanted, in three cases to replace a metacarpal which had to be removed for a bone cyst or chondroma, and in another case to fill a gap in the lower end of the radius following a radical operation for osteomyelitis. Primary healing occurred in all the cases and the bone survived and developed. No trouble was experienced with the foot from which the metatarsal was removed.

Operative Treatment of Flexion Deformity of the Hip Joint.—Stewart,[38] discussing the treatment of flexion deformity of the hip due to infantile paralysis, says that the operation should be planned to preserve as much muscle power as possible for future use. To this end, in cases in which the tensor fasciae femoris remains active, this muscle should not be divided transversely as in the operation of Soutter, but should be lengthened in its tendinous portion, and the transplantation of the lesser gluteals after the manner of Campbell should be held in reserve as an additional measure.

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37. Rankin, William: Glasgow M. J., December, 1924, p. 385.
38. Stewart, S. F.: California & West. Med. **23**:59 (Jan.) 1925.

[ED. NOTE.—As we understand Soutter's operation, the tensor fasciae femoris muscle is not divided, but is transplanted downward from its attachment to the ilium.]

Reduction in Size of Patella.—Ludloff [39] recommends partial excision of the patella in cases of arthritis deformans when the inferior surface, denuded of cartilage and roughened by osteophytes, glides irregularly over the trochlear surface of the femur. In some cases he has excised the central portion, filling the defect with fat. In others he has removed the inferior surface of the bone or he has chiseled away segments 1 cm. thick on either side. The edges were smoothed and in some cases covered with fascia. Distinct benefit was noted in most of the cases.

[ED. NOTE.—Very few cases are seen in which patellar irregularities are the cause of mechanical interference with knee motion. On the other hand, there are many cases of lame knee in which patellar spurs may be revealed by roentgenologic examination. They are not of themselves responsible for the knee symptoms and only exist coincidentally. They are merely the sign of degenerative changes affecting the joint cartilage.]

Operative Correction of Knee Flexion in Poliomyelitis.—Smith [40] describes a procedure for the correction of flexion deformity of the knee due to poliomyelitis. It is indicated only when continuous extension or wedged casts appear insufficient to correct the deformity. He has employed it in fourteen cases, three of which were bilateral. All showed extreme degrees of paralysis. The youngest patient was aged 10 years and the oldest 41. The duration of the paralysis varied from six to thirty-six years. The operation consists of a long incision in the popliteal region, extending from midthigh to the middle third of the tibia. The nerves and vessels are identified and retracted to the outer side of the joint. The inner head of the gastrocnemius is sectioned. The posterior capsule of the knee is divided transversely and the posterior crucial ligament is then sectioned. The latter is most important, as the posterior crucial ligament appears to be the prime deforming factor.

(To be continued)

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39. Ludloff, C.: Zentralbl. f. Chir. **52**:786 (April 11) 1924.
40. Smith, S. A.: Brit. M. J., Dec. 13, 1924, p. 1092.